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DIFFERENTIAL DIAGNOSIS OF VERTIGO *

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NEW ORLEANS

The symptom of vertigo is one of the most common complaints in medical practice, and often the establishment of a definite diagnosis is fraught with considerable difficulty. As confusing as the clinical picture may be at times, the utilization of a definite organized plan of approach to this problem will facilitate diagnosis which is essential for a rational approach to therapy. Unfortunately, the problem of exact diagnosis is not always easily clarified and, to say the least, is sometimes very complex. At the outset, when presented with such a patient, it must be certain that what one is dealing with is true vertigo. If the patient can state unequivocally, after questioning, that it is the sensation one gets after being on a "merry-go-round," or that it is apparent rotation of the field of vision before the patient's eyes, and not fainting or syncope or ataxia of the limbs, then one can be certain that the problem is one of vertigo. Once the examiner is satisfied of this, consideration to its otologic, neurologic, and general medical aspects should be given. The physician, then, must determine whether he is dealing with a problem localized to the ear with its ramifications, brain stem, cerebellum, or with systemic disease.

In order to properly understand the dynamics of vertigo, it is perhaps judicious first to review briefly the anatomy of those structures which play a role in the control and regulation of the sense of balance. The end organ structures of the inner ear, located in the semicircular canals and the utricle are the anatomical units from which emanate impulses which control one's sense of balance. These structures are shown diagrammatically in Fig. 1. They are tubular membranous structures, containing endolymph, located within the hollowed out temporal bone.

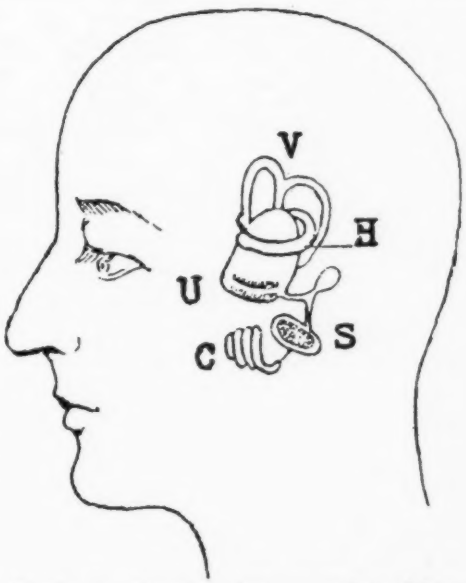

Figure 1. Diagram (not to scale) giving a lateral view of the internal ear within the skull. V, vertical canals; H, horizontal canal; U, utricle; S, saccule; C, cochlea (redrawn from Quix).

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There are three semicircular canals, two of which are vertical and perpendicular to one another, and one which is horizontal all of which represent three different planes of direction. The three are joined at a common end to form the utricle. Within the expanded end of each semicircular canal is the crista on which are located fine sensory hairs which are the nerve receptors. Within the utricle are similar fibers upon which are located the otolith organs. These latter structures are the receptors for the utricle. The cristae are responsible for compensating movements of the eyes and extremities, and the utricle for righting reflexes and compensating positions of the eyes. The semicircular canals respond to changes in velocity and changes in direction.

Impulses emanating from the labyrinth are conveyed to the medulla by way of the vestibular portion of the eighth cranial nerve and the cell bodies of these nerve fibers are located within Scarpa's ganglion. These fibers, illustrated in Fig. 2, pass to the vestibular nucleus in the medulla from which relay fibers follow three pathways:

1. The ascending fibers which join the medial longitudinal bundle of both sides and pass to the nuclei of the third, fourth, and sixth cranial nerves. Through these pathways, reflex movements of the eyes are brought about.

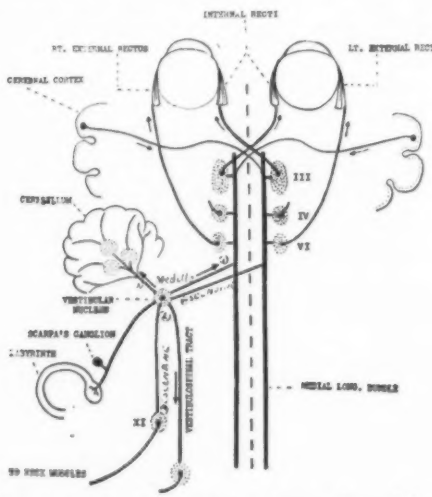

Figure 2. To limb and trunk muscles. Diagram of vestibular pathways.

2. The descending fibers, constituting the vestibulo-spinal tract, connect with spinal motor neurons which carry impulses to skeletal muscle and are responsible for reflex balance.

3. Vestibulo-cerebellar tract, which is the pathway connecting the vestibular nuclei with the cerebellum.

Impulses conveyed over these tracts and others to the cerebrum, by way of the cerebellar peduncles, give rise to the conscious feeling of vertigo.

Abnormal vestibular reactions and sensations then may be evolved as the result of pathological processes within the labyrinth itself, the vestibular nerve, the vestibular centers as Deiters' nucleus (vestibular nucleus), the medial longitudinal bundle and the cerebellum.

The following is a classification of the conditions to be discussed:

I. Lesions of labyrinth
   a. Destructive lesions
   b. Postural vertigo
   c. Meniere's syndrome
II. Lesions of ear outside the labyrinth
   a. Eustachian tube
   b. Middle ear infections
   c. External ear
III. Cerebello-pontine angle lesions
IV. Cerebellar lesions
V. Lateral bulbar syndrome (Posterior inferior cerebellar artery syndrome)
VI. Miscellaneous

I. LABYRINTHINE LESIONS

(a) Destruction of the vestibular apparatus on both sides produces constant unsteadiness of station and gait which is exaggerated by darkness and sudden movements of the head. There is also a striking inability to fix upon objects with the eyes when the head is in motion, so that objects seem to oscillate and dance before the eyes when walking or riding. When the head is kept perfectly still this oscillation ceases. These phenomena are due to loss of vestibulo-ocular reflexes

which influence the extraocular muscles in compensating for movements of the head. A sudden destruction of one labyrinth will cause these symptoms for a few months but in time a compensating process results in disappearance of symptoms. A gradual destruction of one labyrinth will not cause these symptoms. When both labyrinths are destroyed, these symptoms persist indefinitely. Some of the causes of this condition are central nervous system syphilis with involvement of the eighth cranial nerve, bilateral suppurative labyrinthitis secondary to either meningitis or mastoiditis, section of vestibular nerves for Meniere's syndrome, excessive streptomycin, and eighth nerve neuritis complicating mumps or herpes zoster. The labyrinth may also be destroyed functionally by occlusion of the vascular supply to the labyrinth as result of arteriosclerosis or syphilis, or there may be destruction as a result of hemorrhage as in blood dyscrasias or as a result of concussion of the ear. Fractures of the temporal bone may injure the labyrinth and thus be responsible for vertigo. These fractures generally are associated with bleeding into the external canal, the drum frequently tears, and spinal fluid may drain from the ear. Deafness, vertigo, and tinnitus are often seen in this type of injury. The cochlear neurons and vestibular portions of the eighth nerve are known to be susceptible to toxic influences of many infections, particularly typhus, mumps, and herpes. In herpes oticus there is usually seventh nerve palsy, vertigo, deafness, tinnitus and generally pain over the side of the calvarium. This clinical picture is generally referred to as the Ramsey-Hunt syndrome. The fistula syndrome of the labyrinth may result from a cholesteotoma of the mastoid, congenital syphilis, tumors of the temporal bone which erode the labyrinth. As a result of such fistula formation endolymph may pass out of the semicircular canals with movement of the tympanic membrane. Thus the exertion of positive and negative pressure alternately on the tympanic membrane causes motion of the endolymph and produces the sensation of vertigo. This is known as the fistula sign.

(b) *Postural vertigo.* This has been well described particularly by Ford [2] and DeWeese [3] and is a common condition which is often confused with Meniere's syndrome although there is neither deafness nor tinnitus associated with it. It is characterized by vertigo precipitated by sudden movement of the head. Quick motions are more troublesome than slow movements and vertigo is usually mild and brief, but may be violent and accompanied by nausea and vomiting. When still, the patient is usually free of symptoms and usually the change to the same position will consistently provoke vertigo. Though one suspects postural vertigo, it can be proved by having the patient assume the provoking position, then check for nystagmus, the presence of which proves the diagnosis. The physiologic basis for this condition is so far quite obscure; however, it is thought by some that the semicircular canals are stimulated by certain movements and vertigo represents an exaggeration of the normal reflexes. In this condition all otologic and neurologic examinations are negative. The condition is generally mild; however, it may be so severe as to require bed rest for one or two weeks and mild symptoms may recur for several months.

(c) *Meniere's syndrome.* This condition has been well described by many. It is characterized by a sudden violent, spontaneous discharge of impulses from the vestibular apparatus which recurs periodically over a period of years. The onset is abrupt, with severe vertigo, nausea, vomiting, and unsteadiness to the point where one is unable to stand without support. The active phase of such an attack lasts for several hours but the effects of the attack may be felt for several days. Vertigo develops spontaneously, regardless of the patient's movements, and is not relieved by lying still. Between attacks there is rarely a disturbance of equilibrium. One finds in addition to vertigo, tinnitus and deafness. Deafness runs a slow progressive course with

fluctuations in severity, but rarely does total deafness result. One or both ears may be affected. Caloric testing in this condition usually reveals a hypoactive reaction. The fundamental basis for this syndrome is unknown; however, enough histological data has been accumulated to indicate that the pathological process is one of hydrops with dilatation of the endolymphatic system of the labyrinth.

II. LESIONS OF THE EAR OUTSIDE THE LABYRINTH

(a) *Block of the eustachian tube.* In this condition there are changes taking place in reference to pressure within the ear system which stimulate the labyrinth, and thus, the statokinetic system leading to vertigo. Catheterization of the eustachian tube will quickly disclose this process.

(b) *Acute middle ear infection.* Here, due to accumulation of purulent material, an increase in pressure within the middle ear is produced which is transmitted to the semicircular canals, whereby there is stimulation of the statokinetic system. Examination of the middle ear together with other symptoms and signs will disclose the diagnosis. In addition to the above mentioned lesions of the eustachian tube and middle ear, foreign bodies and excessive cerumen in the external ear have been known to produce abnormal stimulation of the semicircular canals, and thus vertigo. However, this is seldom seen as a cause for vertigo.

III. CEREBELLO-PONTINE ANGLE LESIONS

The acoustic neuroma, which is the most common of these, is one which involves the eighth cranial nerve and adjacent structures. The clinical picture, once it has become fully developed, is characterized by:

1. Vertigo and nystagmus with the quick phase of nystagmus towards the affected side.
2. Conjugate paralysis of eyes with deviation to the affected side. This is due to regional edema and pressure with the effect on pontine ocular centers.
3. Hypesthesia over the distribution of the trigeminal nerve which is due to damage to the pons and medulla.
4. Peripheral seventh nerve paralysis with asymmetry of the face.
5. Eighth nerve deafness and tinnitus.
6. Absent caloric reactions on the affected side.
7. Adiadochokinesis.
8. Papilledema, which, as a rule, is late in its appearance.
9. Ataxia of the cerebellar type.
10. Suboccipital headache which is unilateral as a rule.
11. X-ray films of the skull showing widening and erosion of internal auditory canal.

The eighth nerve and cerebello-pontine angle area may also be affected by other tumors, by suppurative meningitis and localized abscesses which may produce in addition to the picture of sepsis a number of the above mentioned characteristics of an acoustic neuroma.

IV. CEREBELLAR LESIONS

Excepting ataxia, vertigo is the most frequent manifestation of cerebellar disease. Cerebellar vertigo is closely related to, or identical with labyrinthine vertigo, for it is a symptom truly of vestibular disturbance and is due to irritation of the vestibular fibers as they course to the cortex of the cerebellum. The vestibular tract takes a course from the labyrinth end organ to the vestibular nucleus, then to the cerebellum.

In cerebellar lesions, while vertigo may be present, the cardinal disturbance is lack of coordination. This means that the various muscle groups fail to collaborate normally in strength, time and speed. This is well disclosed with observations of walking or standing. They have a tendency to fall to the affected side; or, forward or backward in lesions of the vermis. Patients with cerebellar lesions also often demonstrate hypotonia, which is illustrated by having them exert counterresistance of an extremity. When resistance is suddenly released the extremity jerks quickly away and is not checked until the resistance of the joints and ligaments comes into play. In the normal person this motion is quickly checked by

the normal tone of muscles. These patients also have ataxia which is demonstrated by the well known finger-to-nose, heel-to-shin and pronation-supination tests.

Nystagmus is nearly always present in cerebellar disease. There is usually a quick and slow phase; the quick phase being more marked when the gaze is directed toward the side of the lesion. The slow phase is a manifestation of hypotonia and inability to hold the eyes in the position voluntarily moved. Speech is also affected, since it is dependent upon coordinated contractions of muscle groups. As a result of cerebellar lesions then, the voice may be monotonous, drawling, syllables separated and jerked or perhaps slurred. In expanding lesions of the cerebellum, headache is often present together with vomiting as a result of obstruction of flow of the cerebrospinal fluid. Intracranial pressure increases rapidly, papilledema occurs early, and diplopia may occur as a consequence of injury to the sixth nerve.

V. POSTERIOR INFERIOR CEREBELLAR ARTERY SYNDROME (LATERAL BULBAR SYNDROME)

This syndrome is not uncommon, yet it is often misdiagnosed. It is due to a thrombosis of the posterior inferior cerebellar branch of the vertebral artery. As a consequence of its occlusion, a wedge shaped section of the lateral part of the bulbous medulla becomes deprived of its blood supply. With degeneration of this area, a clear-cut clinical syndrome appears characterized by involvement of the sympathetic, trigeminal, glossopharyngeal-vagus nerves, cerebellar pathways, and occasionally the abducent, facial and acoustic nerves homolaterally. The spinothalamic tract is involved contralaterally due to the decussation in the cord inferiorly. The clinical manifestations are (1) pain or dysesthesia on the contralateral side of the trunk and limbs; (2)nystagmus; (3) Horner's syndrome on the affected side; (4) paralysis of palatal arch on the affected side; (5) hoarseness from homolateral paralysis of the vocal cords; (6) ataxia and hypotonia of the homolateral side; (7) vertigo; and (8) difficulty in swallowing.

This syndrome, if thought of and duly considered, is so characteristic that it should pose no problem in diagnosis.

VI. MISCELLANEOUS

There are a certain number of other conditions, too, that should be considered in the differential diagnosis of vertigo. They seldom cause true vertigo but because of the disturbance they produce, which may be confused with vertigo, they deserve mention. In tabes dorsalis and peripheral neuritis, with the loss of proprioceptor nerve fibers, the sense of balance is lost in a fashion, but there is no apparent rotation of objects in space. This loss of balance emanates from the ataxia of the limbs rather than the conscious feeling of vertigo. Postural hypotension, aortic stenosis and Stokes Adams syndrome produce syncope rather than vertigo and should not be confusing if the patient is adequately examined and a good history taken. Thorough history taking and complete examination will eliminate such other conditions as petit mal, glaucoma and multiple sclerosis.

REFERENCES

1. Lindsay, J. R.: Wisconsin Med. J. 49: 607, 1950.
2. Ford, F. R.: Bull. J. Hopkins Hosp. 87: 299, 1950.
3. DeWeese, D. D.: Laryngoscopy 62: 362, 1952.
4. DeWeese, D. D.: J.A.M.A. 142: 542, 1950.
5. Lewis, M. L.: N. O. Med. & Surg. J. 104: 161, 1951.
6. Meriweather, L. S., Culpeper, W. S., and Tuthill, S. W.: M. Clin. N. America, (Mar.) 1952.
7. Best, C. H., and Taylor, N.B.: Physiologic Basis of Medical Practice, 3rd ed. 1943, Baltimore, Williams & Wilkins.

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GALACTOSEMIA IN AN EIGHT DAY OLD INFANT

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A review of the literature on congenital galactosemia reveals that to date only 30 some odd cases have been reported. This condition was first reported by Von Reuss in 1908, although the first clear account

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was given by Groppet in 1917. The majority of these 30 odd cases were not reported until after 1945. The first case to be recorded in American literature was by Mason and Turner in 1935.

Galactosemia is the most amenable to therapy of all diseases of the inborn errors of the metabolism group. Early recognition of this disease and the institution of proper therapy may prevent blindness, mental retardation, and even death.

As reported, (by Edmonds et al [1]), the cardinal symptoms and findings in the congenital galactosemia syndrome are as follows: galactosemia, galactosuria, albuminuria, malnutrition, hepatomegaly, jaundice, mental retardation; and cataracts may occur in various combinations in a given case. A familial tendency has been shown to exist in a recent report by Cox and Pugh [2] of Great Britain which tells of three families with multiple siblings having the disease. Although the number of cases reported to date is admittedly small, this may not be as rare a disorder as was once thought. Many infant deaths signed out as infantile diabetes, hepatomegaly of unknown etiology, jaundice of unknown etiology, and chronic malnutrition may have in reality been the congenital galactosemia syndrome.

The object of this paper is to present another case of congenital galactosemia syndrome, to point up the clinical picture and findings by which the diagnosis can be suspected, and to describe some simple tests to establish this diagnosis. The usual method for identification of galactose as the reducing sugar in the urine is by paper chromatographic method, but since this is not available to most small hospitals a plan for differentiating reducing substances in the urine, with the use of only Benedict's solution, a water bath, and yeast is submitted. A simple method for following the efficiency of the dietary treatment is also pointed out.

CASE REPORT

S. K., eight days old, admitted to the United States Naval Hospital, San Diego, California, on September 14, 1954. Discharge date: September 28, 1954. Admission weight: 7 pounds, 15 ounces.

Birth History: Normal spontaneous delivery after eight hours' labor. Mother's blood type AB, Rh positive. Father's blood type A, Rh positive. No cyanosis or convulsions noted in early neonatal period.

Prenatal History: Mother had no viral diseases during her gestation period.

Early Feeding History: Infant was fed on a formula of evaporated milk, one ounce, boiled water two ounces, white Karo one teaspoon. The infant nursed well but was observed to vomit at about every third feeding.

Birth Date: September 6, 1954. *Birth Weight:* 9 pounds, 6 ounces.

Present Illness: The baby had been vomiting since birth and for the past forty-eight hours prior to admission had begun to show progressive lethargy. The parents noted that a yellow color to the skin and eyes had developed on the fifth day of life. No other history was pertinent.

Family History: This was the fifth pregnancy. The first born child was delivered in Spokane, Washington on May 30, 1948. Birth weight was 7 pounds, 12 ounces. At five days of age the child developed vomiting, loose stools, and became progressively jaundiced. At eight days of age the child was taken to the family doctor who made a diagnosis of "enlarged liver". A medication was prescribed to be given every three hours. After having showed no improvement at eleven days of age, the infant was taken to the United States Naval Hospital at Oakland, California. His admission diagnosis there was dehydration and acidosis. When routine urine analysis showed sugar to be present, this diagnosis was changed to infantile diabetes. Although insulin and many other medications were prescribed, the infant continued a downhill course until he expired at three months of age with the body weight of 6 pounds, 8 ounces.

The second child was a male, from a full term pregnancy on January 9, 1950, who has progressed well to date.

The third child was a male, from a full term pregnancy on January 24, 1951; who progressed well to date.

The fourth pregnancy resulted in a stillborn at five months' gestation.

The paternal grandmother states that twenty-nine years ago she lost two children at ages four weeks and six weeks who became jaundiced soon after birth, vomited, were listless and failed to gain weight. She had three other normal children.

Physical Examination: Temperature—98.6°F. (R). Pulse 120. Respirations 18. Well developed, well hydrated eight day old female infant, whose skin and eyes reveal icterus.

H.E.E.N.T. Negative. Careful examination of eyes revealed no cataracts.

Neck: No masses, supple.

Chest: Bilateral equal excursions, no gross abnormalities noted.

Lungs: Clear to percussion and auscultation.

Heart: Normal sinus arrhythmia. Not enlarged. No murmurs audible.

Abdomen: Flat. Liver down 3 centimeters. Kidneys and spleen not palpated. No masses noted.

Extremities and Genitalia: Normal.

A complete neurological examination was negative.

Laboratory Reports:—The following data were obtained while the infant was on a lactose containing diet.

1. Hemogram: R.B.C. 5,300,000. Hemoglobin 16 grams. W.B.C. 8,800; neutrophiles 48%; lymphocytes 52%.

2. Urine tests: Albumin, 150 mgs. per 100 c.c.; sugar 2 plus positive to Benedict's test; Bile negative; Urobilinogen, 1:20 dilution positive.

3. Van Den Bergh Reaction: Direct 5.9 mgs. percent; indirect 14.5 mgs. per cent. Total serum bilirubin level 20.4 mgs. per cent.

4. Nonfasting blood sugar level: 210 mgs.

5. A blood galactose level was set up and performed as follows: 1 c.c. of the patient's oxalated blood was incubated with 1 c.c. of a 10 per cent suspension of washed yeast for ninety minutes. Two controls were incubated under the same conditions. Blood sugar levels were obtained on the samples both prior to and after incubation with added yeast.

Results: Sugar concentrations on the non-incubated specimens—

Patient's blood: 210 mgs. Control A: 70 mgs. Control B: 130 mgs.

Sugar concentrations on the yeast incubated specimens:

Patient's blood: 81 mgs.

Control A: 5 mgs.

Control B: 10 mgs.

From these results it is apparent that approximately 92 per cent of the dextrose in controls A and B was hydrolysed. Assuming that this is also true for the patient's blood, then the blood galactose level is 71 mgs. or $\frac{210\text{-}81}{0.92} = 139$ mgs. of dextrose. 210-139 equals 71 mgs. of galactose.

6. A large amount of urine was collected while the infant was on a lactose containing formula and the following plan was set up to identify the reducing substance in the urine. This technique is outlined in Ham's *Syllabus of Laboratory Examination in Clinical Diagnosis.*

Course: When the above tests were completed, the infant was placed on Sobee milk.* Within forty-eight hours all urine specimens reacted negative to Benedict's urine sugar test. The infant began to nurse well and the jaundice subsided until on September 28, 1954 the infant was discharged from the hospital weighing 9 pounds, 8 ounces.

* A product of Mead Johnson Company.

ORIGINAL URINE SPECIMEN

Benedict's test (boiling, five minutes)

If **negative**, then no reducing substances.

If **positive**, then repeat Benedict's on sample of original specimen at 55° C. for ten minutes.

If **negative** the possibilities are:	If **positive** the possibilities are:
1. Glucose under 4%	1. Pentose
2. Maltose	2. Fructose
3. Lactose	3. Glucose 4% plus
4. Galactose	
5. Non-sugars	
On 30 ml. of the original specimen do; **Fermentation test** (which removes glucose and maltose.) When fermentation is completed then repeat on this specimen the Benedict's (Boiling, 5 minutes):	On 30 ml. of the original specimen do; **Fermentation test** (which removes glucose and maltose.) When fermentation is completed then repeat on this specimen the Benedict's (Boiling, 5 minutes):
If **negative** the possibilities are:	If **positive** then pentose is present.
1. Glucose under 4%	
2. Maltose	
If **positive** the possibilities are:	If **negative** the possibilities are:
1. Lactose	1. Fructose
2. Galactose	2. Glucose 4% plus
3. Non-sugars	To differentiate do Seliwanoff's test on a sample of the original specimen:
(Rubner's and phenylhydrazine tests may now be of specific value.)	Fructose gives a positive reaction, while glucose gives a negative reaction.

By this means the reducing substance in the patient's urine was identified as galactose.

Follow Up: The infant was seen by the author in the clinic on October 12, 1954. He had done well and there was no evidence of any jaundice. The liver was only barely palpable. Careful examination of the eyes failed to reveal any evidence of cataracts.

DISCUSSION

Once one is familiar with this syndrome it should offer little diagnostic difficulty. Any infant who in the early newborn period manifests listlessness, vomiting, failure to gain weight, jaundice, and hepatomegaly should have a urine examination. The presence of albumin and a positive sugar reaction should then provide a high index of suspicion for the congenital galactosemia syndrome.

Pratz found in an extensive review of 25 of the reported cases of this syndrome that 19 had cataracts present.[3] Although the above presented case had no evidence of cataracts, this is described as an important feature of this syndrome. The most commonly observed opacity appears as a central refractile ring resembling the "oil drop" effect. Clusters of cortical vacuoles and various forms of zonular cataracts have been described. In view of the rapid cataractogenic effect of galactosemia, periodic examinations by an ophthalmologist should be a part of the management of these cases.

Treatment of these cases can be summed up in one sentence: The exclusion of lactose containing food from the diet. Sobee, made by the Mead Johnson Company, is excellent for diets of these infants. As new foods are added with progressive age, it should be borne in mind not to add other lactose containing foods such as custards and butter. Oleomargerine is an excellent substitute for butter. As the child grows older, Townsend[4] has stated that some milk containing food may be added with no adverse effects. An excellent rough test for the efficiency of the dietary treatment is to have the mother perform urinary sugar tests with the sugar test tablets.† These tests should be done a minimal of twice weekly and any positive results should be reported to the physician at once.

Without treatment most cases will die. Even with early treatment prognosis should be guarded. In the case reported by Townsend et als[4] in 1951, a child was begun on treatment in the first few days of life, yet this child is now thought to be mentally defective. Mental retardation is a great hazard in these cases.

SUMMARY

A case of galactosemia in an eight day old infant has been presented along with laboratory methods for galactose identification in urine and blood. The possibility of galactosemia should be considered in any infant who fails to gain weight, who may or may not show vomiting and jaundice, who has hepatomegaly, and may or may not show cataracts. A urine analysis should be obtained. The presence of albuminuria and sugar should offer a provisional diagnosis. Early diagnosis and early treatment may prevent mental retardation, blindness, and even death. Treatment is best followed by frequent urinalysis. Prognosis should be guarded.

The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the U. S. Navy Department or the Naval Service at large.

REFERENCES

1. Edmonds, A. M., Hennigar, G. R., and Crooks, R.: Galactosemia, Pediatrics 10:40, 1952.

2. Cox, P. J. W. and Pugh, R. J. P.: Galactosemia, Brit. M. J. 613, 1954.

3. Pratz, A.: Cataracts in galactosemia. Am. J. Ophthalmol. 36:453, 1953.

4. Townsend, E. H., Mason, H. H., and Strong, P. S.: Pediatrics 7:700, 1951.

† A product of the Ames Company.

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LIVER DISEASE IN AMBULATORY PATIENTS *

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NEW ORLEANS

"Unfortunately, only the advanced stages of hepatic injury are easily recognizable. . . . Even when liver disease is sus-

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pected, symptoms frequently are not sufficiently well-defined to indicate a definite diagnosis. We have swung too far from the old tendency to ascribe a variety of ill-defined complaints to 'sluggishness of the liver'. To deny its role in illness unless frank jaundice, ascites, circulatory interference, or disturbance of clotting time unmistakably point to that organ, is to take too narrow a view of the importance of the liver." This quotation from a Lederle Bulletin, May 1950, author unknown, very aptly expresses the theme of my presentation.

In recent years great progress has been made in the recognition of diseases of the liver. Liver function is measured by many sensitive tests from which the physician may choose those best suited to the problem under consideration. Liver biopsy has proved a useful tool in selected cases. The vast amount of current knowledge relating to liver disease has necessitated the publication of monographs in this field, and has created a new medical specialist, the hepatologist. However, current texts and articles do not describe or consider incipient, reversible, and mild liver disorders which we encounter among office patients presenting themselves with gastrointestinal symptoms. The disorders to be described do not at present have an acceptable medical name. Let us consider the means by which these conditions are recognized.

First, liver disease may be recognized by certain combinations of symptom patterns. During the war years, experience with the various types and stages of hepatitis indicated that during an epidemic of viral hepatitis one could suspect and confirm the diagnosis of hepatitis without jaundice.

At the beginning of one outbreak soldiers were observed who had the early symptoms of acute hepatitis; anorexia, nausea, and upper abdominal discomfort. They then went on to develop jaundice of only a few day's duration. A study of the relative frequency of the symptoms observed in liver diseases is presented. (Table 1).

TABLE 1
SYMPTOMS OF LIVER DISEASE

Clinical Conditions Considered

Symptoms of primary parenchymal defects liver (Lichtman)
Pre-icteric symptoms acute hepatitis
Acute hepatitis without jaundice
Chronic viral hepatitis
Sequelae of acute hepatitis
Early symptoms portal cirrhosis

Frequency of Symptoms

Anorexia	6	Myasthenia	1
Fatigability	5	Hepatic coma	1
Diarrhea	4	Giddiness	1
Epigastric discomfort	4	Lassitude	1
Nausea	3	Hepatic tenderness	1
Rt. U.Q. Ache	3	Nervousness	1
Vomiting	3	Malaise	1
Arthralgia	3	Depression	1
Dyspepsia	3	Flatulence	1
Weakness	2	Headache	0
Fatty food dyscrasia	2		
Myalgia	2		

The conditions considered are: pre-icteric symptoms of acute hepatitis, acute hepatitis without jaundice, chronic viral hepatitis, the sequelae of acute hepatitis, the early symptoms of portal cirrhosis, and symptoms described by Lichtman [1] as the result of primary defects in the liver parenchyme. Before one can assess any diagnostic evaluation of symptoms, he should be familiar with the total patient; his somatic and psychosomatic components. Previous knowledge of the behavior and medical background of the patient over a number of years is desirable. The physician in general practice is therefore in a better position to evaluate his patient in this regard. One does become familiar with the usual display of symptoms in his psychoneurotic patients. A positive diagnosis of neurosis should be made rather than diagnosis of this condition by exclusion. Food allergy should be considered and excluded, and of course the possibility of other gastrointestinal conditions must be eliminated.

In connection with the symptomatology of reversible liver conditions, it is of interest to consider the meaning of the term "bilious". Bockus [2] states that the former usage referred to episodes of headache, nausea, and vomiting which physi-

cian and patient attributed to the gall bladder. Many of these were attacks of migraine and were not caused by the gall bladder or stones if these were present. To Burril Crohn [3] the term "biliousness" implied symptoms which in a measure were the result of intestinal auto-intoxication. There have been many other names previously applied to disorders akin to or connoting the same as "biliousness". Levin [4] used the term hepatic toxemia. Older writers used the term hepatism. Hurst [5] called these patients "liverish". Into this cateory were placed the vague liver symptoms of tropical chronic malaria. Lichtman [1] uses the term hepatic insufficiency which might be applicable here. Willcox [1] has coined the term hypohepatism. Portis [5] used the term subclinical hepatitis to apply to the patient who was alcoholic with a fatigue state. These examples serve to focus attention on a group of patients one encounters in practice. None of the aforementioned terms have gained acceptance. We propose use of the term "hepatosis". Let us examine the scope of this clinical concept. Hepatosis may be acute or chronic. There may be no symptoms other than an enlarged liver. Other cases present symptoms which may be associated with parenchymal liver involvement. In about one half the cases some degree of enlargement of the liver may be detected. Nearly all show increased amounts of urobilin or urobilinogen in the urine.

Many of these patients give a history of a high fat diet and are addicted to gravies and fried foods in excess. Others simply overeat. The use of liberal amounts of alcohol and a frequent incidence of allergy are also noted. The point should be made that it is believed that these conditions are reversible, for the most part. Follow-up over a long period of time or the use of liver biopsy would probably indicate that some of them are latent cirrhosis. Consideration of the pathology is purely speculative. The acute episodes might be expected to show increased liver size and weight by virtue of increased amount of fluid or blood in or between the liver cells. It has been shown that the young liver is capable of marked variations in size in a very short period of time depending upon the status of the vascular components of the liver. Other cases might be expected to show a fatty liver which frequently is noted when liver biopsy is done.

ENLARGEMENT OF THE LIVER

The detection of liver enlargement is another means of recognizing liver abnormalities in ambulatory patients. The size and weight of the liver depend upon the amount of fluid and blood it contains. Dramatic excursions in liver size occur in twenty-four hours. Rapid shrinking of the liver has been noted in acute infections treated with antibiotics. We have noted sudden enlargement following a huge meal of food and drink which taxed the functional capacity of this organ. Increase in size of the liver may be produced by increase in the fluid content, fat content, or enlargement of the cells of this organ. Palpation of this organ still remains the best clinical method for detecting enlargement. Osgood and Habbe [6] feel that the palpability is affected by the firmness of the liver substance and that some enlarged organs remain undetected. However, attempts to measure liver size by x-ray have not been successful. No good correlation has been obtained between the size of the liver x-ray shadow and the ability to palpate the liver edge. The many surfaces and borders make it almost impossible to measure the liver volume by x-ray. Radiography might prove clinically useful in studying reversible liver disorders where diminution in size of the organ would be expected following therapy. It is practical to secure a film after marking the edge of the liver as palpated with a lead strip marker. A second film using the same exposure factors could then be made following a suitable period of therapy when the liver has resumed its normal proportions. Certain clinicians have felt that palpation of the liver yields unreliable results. Rives,[7] in 1937, reviewed the findings in 96 patients with carcinoma of the pancreas.

Fifty-one per cent of 41 autopsy cases were said to have a palpable liver. Only 21 per cent were found to be enlarged at autopsy. In this series, 26 cases showed enlargement of the liver which was not noted clinically. Armaz-Cruz,[8] in 1951, found that in 43 autopsied cases of portal cirrhosis with clinically enlarged livers only 20 had a real increase in weight. These findings do not alter the fact that palpation of an enlarged liver is clinically significant. Clinicians continue to use this method. The accuracy of recorded findings in a large general hospital is open to some question. In both series there was no separation of cases which presented a liver edge which was barely palpable. This finding should not be considered abnormal. Another reason to make us question the validity of these observations is the experience which we have noted regarding the size of the liver as observed post-mortem. A liver edge palpable repeatedly during life as greatly enlarged is found at the post-mortem table to be much smaller insofar as the edge is seen to protrude below the costal margin. The weight of such a liver did not correllate with the size as observed during life. The reason for this is that the size of the liver changes after death depending upon the weight of fluid and blood remaining in the organ. Palpation of the liver by Ricketts and Kirsner[9] in 50 cases of latent portal cirrhosis revealed hepatomegaly in 44. The detection of an enlarged liver in this series was the key finding which led to the recognition of this abnormality. The final point which should be made is that palpation of a liver edge two or more fingers breadth below the costal margin in the right midclavicular line can be considered evidence of hepatomegaly because we have repeatedly seen it return to normal size following therapy.

LIVER FUNCTION TESTS

There are more methods of testing liver function today than there are functions of the liver. We will omit from consideration the battery of useful liver tests which may be employed in the study of hospital cases. Let us consider those which may be employed in the study of ambulatory hepatic patients. The tests are simple enough to be performed in a doctor's office. Those used in this study were the following:

1. *Bromsulfalein Test.* In the past many values have been proposed as the upper limit of normal for this test. There has been no uniformity in the size of the dose given, the time of collection of specimens, and the expected normal variation. At present, acceptance of the 5 mg. per kilo dosage is general. Dye retention is measured after a forty-five minute interval, and retention of greater than 5 per cent is considered abnormal. This test gives positive results where liver damage is diffuse and extensive. It is one of the sensitive screening tests to detect early liver involvement. It is particularly useful in detecting cases of latent cirrhosis. It is of interest to consider the significance of borderline values. With the use of a photo-electric colorimeter to measure dye retention, minor variations and degrees of dye retention may be perceived. In our office patients nearly all show values under 2 per cent retention at the end of forty-five minutes. We watch more carefully the future course of people with suspected liver disease with values near the upper limit of normal. A stress situation, such as surgery, may convert a borderline bromsulfalein retention into one which is definitely abnormal.

UROBILINOGEN IN THE URINE

The significance of urobilinogen in the urine was called to our attention years ago by the late Foster Johns.[10] Later at the bedside we observed the material upon whom Allan Eustis [11] of this city conscientiously ran urinary urobilinogen determinations. He reported, in 1927, 15,000 urine examinations. He found that 6 per cent showed a 3 plus reaction. An additional 23 per cent were 1 and 2 plus. He noted the association of positive tests with indicanuria. His figures are somewhat difficult to compare with other series since the test was performed by boiling the urine specimen. He attached

little significance to a 3 plus reaction or a 1:150 Wallace diamond reaction. Today a positive 1-40 is considered abnormal. The best methods for office tests are the Wallace diamond technic with Ehrlich's aldehyde reagent or the determination on a 2 to 4 p.m. urine specimen following the quantitative procedure as outlined by Watson.[12]

The test for urobilinogen in the urine in recent reports proved its usefulness by detecting early viral hepatitis (Swift 1950),[13] and by its results in portal cirrhosis. Rickets[9] found 30 per cent of asymptomatic portal cirrhosis positive by this method and 70 per cent positive in advanced cases. It has been our procedure to rule out porphyrinuria in all urine specimens positive for urobilinogen.

UROBILIN IN THE URINE

Comparatively little attention has been paid to the presence of urobilin in the urine. The current concept favors Watson[1] who feels that urobilinogen is only formed in the intestinal tract as a reduction product of bilirubin. There are several forms of urobilinogen, namely, mesobilirubinogen and stercobilinogen. The first, mesobilirubinogen, oxidizes to produce urobilin. It has, therefore, been the opinion that urobilin forms in old urines from urobilinogen and that the chemical would not be present in freshly voided specimens. We have found that the addition of Lugol's solution to such urine many times results in the demonstration of abnormal amounts of urobilin when urobilinogen is not present. The test for urobilin is performed by first precipitating any bilirubin present with 10 per cent calcium chloride. To the filtrate is added a few drops of Lugol's solution and a saturated solution of zinc acetate in alcohol. After centrifuging, the tube is examined in the beam of a microscope lamp for green fluoresence. Factors which we have found giving rise to a false positive test are: (1) Recent previous administration of vitamin B. (2) Delay in reading the test. An additional thirty minutes may convert a negative to a positive. (3) The addition of an excess of zinc acetate in suspension. (4) Bilirubin in the urine unless first precipitated by calcium chloride. Another fact is that a strong test gives a pink-orange color when viewed by direct light and a green fluoresence when viewed by indirect light. We have considered any definite green fluoresence to be a positive test. The amount of urobilin in the urine may be estimated by determining the greatest dilution which will still result in a positive reaction. We feel that normal individuals do not show a positive test for urobilin in the urine and that the interpretation of a positive test is like that for urobilinogen. Urobilinuria denotes liver dysfunction.

The other liver function tests considered desirable in the study of ambulatory liver patients include: Thymol turbidity test, cephalin cholesterol flocculation test, bilirubin in urine-diazo spot test of Godfried.

STATISTICAL DATA

The first group of cases reviewed includes 195 cases studied in the office on whom, among other studies, a urine test for urobilin was performed. In about half of these cases liver disorders were suspected. They do not represent a consecutive group of new patients. Follow-ups are available on the majority.

Of this group of 195 cases urobilinuria was present in 95; both urobilin and urobilinogen were found in 113. Of the entire 195 cases, 66 were felt to have definite biliary tract disease; 37 showed questionable liver disease; and 92 had no liver or biliary tract disease. In the 95 patients with urobilin in the urine final diagnoses of definite liver disease were made in 60 cases, while 22 had questionable liver disease, and 13 had no liver disease at all.

Of 66 cases which had definite symptoms of liver disease, 28 had hepatosis, 18 had fatty livers, 10 cholelithiasis, 6 acute hepatitis, 1 chronic hepatitis, 2 heart failure, and 1 had malaria. Thus, these figures point out that we encountered with greatest frequency a type of liver disorder not described in current

texts or monographs.

It might be of interest to review the incidence of allergy in this series. Of 113 cases showing urobilin or urobilinogen in the urine 17 were allergic, 82 cases were negative, and 14 showed evidence of clinical allergy.

In order to correct any impression that a positive test for urobilin in the urine is found in a large proportion of patients studied in the office another series of our cases was reviewed. In this recent group of cases the test was performed routinely on all new office admissions, the majority of whom had gastrointestinal complaints. In 100 cases a positive urobilin was found in 17. The chemical nature of the material in the urine giving a positive test was studied by precipitating bilirubin before testing for urobilin and by performing a spot test on the urine for bilirubin by the Diazo spot test of Godfried.

CASE REPORTS

It may be of interest to record briefly several types of clinical pictures encountered in hepatosis eliminating all irrelevant material. These patients were followed over a long period of time and were completely studied.

Case No. 1. Following a sumptuous meal of superabundant food and drink at a famous French New Orleans restaurant, a young male physician was examined the following morning. Nausea, giddiness, and pain in the right upper quadrant were present. An extremely tender liver edge was felt 3 fingers' breadth below the costal margin. Urine urobilin was strongly positive. The symptoms and enlargement of the liver lasted only twenty-four hours. Follow-up over a period of years showed no recurrence of symptoms.

The initial response to this case was to question what the pathology might be and what the liver biopsy might be expected to show. It is felt that the only pathology here is engorgement of the liver accounting for the sudden marked variations in size. The disturbed physiology is probably related to the delayed gastric emptying time expected after the ingestion of a large fatty meal with alcohol. The liver was probably working overtime to accomplish its mission in digestion.

Case No. 2. E. B., a white male of 52, was observed over a period of years with abdominal pain resulting from food allergy. In 1953, he was seen complaining of frontal headache after eating watermelon. The liver was found to be 2 fingers' breadth enlarged. Urinary urobilin was positive. The bromsulfalein showed 0.08 per cent retention at the end of thirty minutes. The headache promptly cleared and liver size was found to be normal five months later.

This case is classified as acute hepatosis which is part of an acute allergic episode involving the liver.

Case No. 3. G. D., white male of 47, presented symptoms over a long period of time. In September 1946, there was weakness, drowsiness, and bloatedness. The urobilin test was positive. In November, he became costive. Urobilin test was again strongly positive. In April, the following year he developed weakness and dyspepsia after a trip to the country in which he consumed much greasy food. In August of that year, he reported dyspepsia after eating seafood. Urobilinogen in the urine was strongly positive. In November 1947, there was arthralgia with a 3 plus urobilinogen in the urine. February 1948, following overwork he reported weakness and dizziness. Urobilin was present in the urine. In April 1953, he stated that he had felt tired for one month. The liver edge was felt for the first time enlarged 2 fingers' breadth below the costal margin. Allergens were eliminated and a low fat diet ordered. One month later all findings were negative. This patient was allergic to seafood, watermelon, milk, cabbage, greasy foods, and tomato gravies by repeated clinical trial. The usual laboratory tests and gall bladder and gastrointestinal x-rays were negative.

This case is of interest because he presented at one time or another all the symptoms of hepatosis, because a definite food allergy was also present, and because evidence of liver enlargement was detected more than six years after the first appearance of symptoms.

Case No. 4. The following is reported to illustrate an asymptomatic chronic hepatosis. A white male upon routine physical examination was found to have a liver edge 2 fingers' breadth below the costal margin. He was a beer drinker who liked to eat fats, gravies, and fried food. The urine showed urobilin. The blood cholesterol was 319. Total bilirubin was 0.72. No bromsufalein was retained after thirty minutes. Thymol turbidity measured 7 units. All findings were negative after two months on a low fat diet.

Case No. 5. The patient was a white male who presented himself in 1946, stating that he was weak, costive, dizzy, and had a dull headache. A tender liver edge was palpable 3 fingers' breadth below the costal margin. The urine was negative and bromsulfalein normal. The findings cleared with treatment. A year later fatigue and headache recurred. The liver edge was again felt 3

fingers' breadth below the costal margin. During a six year observation period there have been no recurrences. This patient had a typical picture without urinary findings.

Case No. 6. The patient was a 50 year old colored, mildly diabetic, obese male. In 1949, the urine was strongly positive for urobilinogen. There was 3.8 per cent bromsulfalein retained in thirty minutes. In 1952, he was asymptomatic. The B.S.P. was 1.3 per cent in thirty minutes. The urine was strongly positive for urobilin. This year a cholecystogram was done and revealed impaired concentration of dye and a number of small stones. The finding of urobilin and urobilinogen in the urine should make us think also of the possibility of gall bladder disease. Several similar cases have been observed of gall bladder disease existing without indicating its presence by means of biliary colic as a signal.

CONCLUSIONS

1. In daily office practice we see patients with liver disorders not described in the literature.

2. The term hepatosis is proposed to describe these reversible liver disorders.

3. Enlargement of the liver as a diagnostic aid is discussed.

4. The presence of urobilin in the urine is found to be of great significance in the diagnosis of early liver disease.

REFERENCES

1. Lichtman, S. S.: Diseases of the Liver, Gallbladder and Bile Ducts, Lea and Febiger, Philadelphia. 2d ed., 1949.

2. Bockus, H. L.: Gastroenterology, Saunders, Philadelphia. 1943.

3. Crohn, Burrill: Personal Communication.

4. Levin, A. L.: Personal Communication.

5. Portis, Sidney: Panel Discussion, Am. C. Phys. 1953.

6. Osgood, C. W., Habbe, J. E.: A clinical and roentgenologic study of factors influencing the palpability of the liver, Radiology, 21: 66, 1933.

7. Rives, J. D., Romano, S. A., Sandifer, F. M.: Carcinoma of the pancreas, Surg. Gyn. & Obst. 65: 164, 1937.

8. Armas-Cruz R. et al: Portal cirrhosis; An analysis of 208 cases with correlation of clinical, laboratory, and autopsy findings, Gastroent., 17: 327, 1951.

9 Ricketts, W. F., Kirsner, J. B., Kipper, D. D., and Palmer, W. L.: Tests of "hepatic function" in portal cirrhosis, Gastroent., 15: 40, 1950.

10. Johns, Foster: Personal Communication.

11. Eustis, Allan: The clinical value of Ehrlich's Aldehyde reaction, New Orleans M. & S. J., 79: 593, 1927.

12. Watson, C. J., Schwartz, S., Sborov, V., and Bertie, E.: Studies of urobilinogen Simple method for quantitative recording of Ehrlich reaction as carried out with urine and feces, Am. J. Clin. Path. 14: 605, 1944.

13. Swift, W. E., Jr., Miller, W. N., Streitfeld, F. H., Knowlton, M.: Evaluation of 2 screening tests for detection of early and subicteric viral hepatitis, Am. J. Med. 8: 581, 1950.

DISCUSSION

Dr. Murrel H. Kaplan (New Orleans): As one who has been intensely interested for many years in diseases of the liver, I am deeply grateful to Dr. Shushan for bringing out several enlightening and illuminating facts.

First, specialization has really gone far in creating the "Hepatologist". It won't be long before we will see the rise of the right and left "lobulist" and the "interlobulist".

Secondly, during the World War II, we had the opportunity to confirm Dr. Shushan's statement on palpation of the liver as an unreliable index of hepatic enlargement. There were many who felt that a palpable liver was paramount to a diagnosis of hepatitis. We attempted to show that there was no correlation between the palpable liver and the sign by x-ray study: that there was no correlation between the palpable liver and jaundice; and what was more, individual physicians varied in their interpretation of what they felt in the upper right quadrant. All of this still met with the shake of the heads from the late Dr. Barker of Chicago and his colleagues. It was really not until Dr. Roy Turner of New Orleans entered the picture that a more sane attitude was taken toward the palpation of the liver and its relationship to disease.

Third, it is obvious from the work of Dr. Shushan that bile tract dysfunction is just as common as our older physicians claimed. It is unfortunate that in our time, most of us have lost ourselves in the maze of x-rays and highly specialized examinations and have brushed aside the more simple and much more informative tests such as those for urobilin and urobilinogen.

"Hepatosis", as I understand Dr. Shushan, must mean functional or noninfectious biliary disease. I am reminded of a paper by Dr. Wm. Boyd, Professor of Pathology, Univ. of British Columbia, Vancouver, at a recent meeting of the American College of Physicians. In his paper on collagen diseases, he remarked that there were two schools of thought. The "lumpers", those who felt there was a common pathological basis, and the "splitters" who felt that each had a different process. He was a "lumper" so far as collagen disease was concerned. I am a "lumper" in my thoughts on liver disease.

Time does not permit anything but a mere reference to the great work on "Stress" by Dr. Hans Selye. In his book, hepatitis is listed as a disease of adaptation, whose common etiological basis is stress.

These stressor agents, or alarm stimuli, bring out reaction from the body, which we call resistance. This, of course, varies whether it is bacterial, chemical, or functional, but its effect upon a specific target or organ, pathologically, is similar.

In a recent paper on pancreatitis, it was my good fortune to review the fact that the pancreas and the liver were anlages of the duodenum. The implications of their common embryological

basis was mentally stimulating.

Anatomically these organs have essentially the same nerve and arterial blood supply. (I am aware of the portal vein and its course in the liver). Inasmuch as we are more familiar with the diseases of the duodenum, the most common of which is the ulcer, it was felt that with the above information, we were at the threshold of making certain statements regarding liver diseases.

If duodenal ulcer is accepted as a disease of poor adjustment or adaptation, (as most of us have), should not organs, embryologically from a common stalk, having the same nerve and blood supply, be expected to act and react physiologically to certain stimuli in like manner? If we can answer this satisfactorily, we can probably clear up some of the mystery of "hepatosis".

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AUTONOMIC DYSPHONIA *

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Ever since Garcia first succeeded in visualizing his own vocal cords, laryngeal function has been the subject of most careful and detailed study. Anatomists, physicists, otolaryngologists, voice teachers, and engineers, have contributed much to our present day knowledge of the larynx, and have demonstrated the dual function of this organ; namely, in tone production, and as the watchdog of the tracheobronchial air passageway.

As physicians, our training for the recognition and treatment of speech disorders has been strictly limited to the visual approach. We have become thoroughly familiar with the appearance of the vocal cords by either the direct or indirect view. Our understanding of vocal cord function is based only on what we hear in our patient's voices, and what is related to the appearance of the larynx with the tongue forcibly projected. Is this circumstance conducive to true appreciation of the vocal problem? We hear vocal symptoms every day. Are we able to evaluate them fully with our present understanding of laryngeal function? Inertia and willingness to accept time worn theories have fostered ingenious, albeit poorly supported reasons, for disorders of speech.

Since it has been considered that vocal tone is produced solely by the action of the vocal cords, efforts have been constantly made to solve all vocal problems by relating them to the action and appearance of the vocal cords. So many vocal disabilities have been and are still being attributed completely to organic laryngeal pathology, that it behooves us to reexamine some of our long accepted concepts.

The larynx is neither anatomically nor physiologically independent, but must be considered as an integrated part of the human biological system.

The voice plays a singularly important part in human expression. Not only is it influenced by respiration, which is controlled by the autonomic nervous system, but since the voice is a form of emotional expression, it is vulnerable to any pathologic condition which disturbs the emotional life. The act of vocalization is a function of the total personality in its response to varied situational and environmental influences.

While it is important to distinguish dysphonia of pure organic origin from vocal dysfunction of psychosomatic origin, this attempt is usually most difficult. Any strain, any change in vocal range or intensity which surpasses physiological limits, irritates the vocal cords and produces changes in their basic appearance.

At this point, I should like to make it clear that I do not wish to give the impression that I regard all or even most cases of dysphonia to be on an emotional etiology. It is my purpose to stimulate interest in a most frequently occurring phenomenon, whose recognition is based on suspicion and knowledge.

Emotional disturbances, through their effect on the autonomic nervous system, can produce vocal cord dysfunction. Instances of functional nature, as cited later, will demonstrate where personality rehabilitation and reassurance were effectively applied, and the vocal dysfunction ameli-

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orated. It will also be noted, that despite the dearth of organic pathology, variations of interpretation as to the cause of the dysphonia were numerous. The most frequent diagnoses were laryngitis, myasthenia laryngis, vocal nodules, thickened vocal cords, contact ulcer, and others of more or less far flung imagination.

It immediately becomes apparent that there is real need to survey the individual as a whole. Such investigations must include a careful history, a complete examination, and an appraisal of the social, emotional, and economic adjustments of the individual. In our role as physicians with ever broadening horizons, we must at least recognize the presence of individual maladjustments and their ability to produce vocal dysfunction. Rational key therapy can be administered only if such a procedure is followed. It is not only useless, but illfounded to treat these dysfunctions by unnecessary and potentially harmful medical and surgical procedures.

We must consider that the autonomic nervous system has a direct effect on the function of the vocal cords, since the vagus nerves through its laryngeal branches innervate the intrinsic laryngeal musculature. It is my concept that myasthenia laryngis, without generalized myasthenia gravis, and the formation of a certain percentage of vocal nodules and contact ulcers, actually represents progressive dynamic phenomena brought about through the abnormal stimulation of the autonomic nervous system, and its direct effect on the function of the vocal cords. The progression of vocal cord difficulty commencing with symptomatic referral from conflict, to reversible and finally irreversible changes, is comparable to similar findings in the gastrointestinal tract ranging from the peristaltic belly-ache to the irritable mucusproducing colon, to the final stages of ulcerative colitis.

The individuals whom I am about to consider demonstrated anxiety in one form or another far overshadowing their physical state. They were tense, restless individuals, appearing ill at ease, perspiring freely, always fatigued, and usually associated with varying degrees and manifestations of gastrointestinal and cardiovascular difficulties. Visualize the tense, anxious individual with the complaint of tight throat, power lacking strained voice, and above all the irregularities of pitch. The vocal cord manifestations in this stage are sparse, and may only reveal some injection of the approximating margins. These manifestations are primarily symptoms of the patient's emotional tensions and the misdirected efforts to project his voice through laryngeal actions. He utilizes the voluntary musculature of his throat and neck muscles in attempts to increase the carrying power of his voice. All to little avail, for the exaggerated pressure produces tense adduction of the vocal cords with friction which is an inhibitory mechanism. Thus the mechanism of vocal nodules and contact ulcer formation becomes more readily understandable.

In Jackson and Jackson "Diseases of the Nose, Throat and Ear", published in 1945, it is stated that, "The exciting cause of myasthenia laryngis is trauma in the form of vocal abuse as a result of overworking the thyro-arytenoid muscles". Contact ulcer of the larynx is chiefly caused by the hammering of one cartilagenous vocal process against the other, traumatizing the thin mucosal covering. "Vocal nodules are due to singing or speaking out of normal vocal register, or attempting to produce volume of greater than normal capacity."

It is my contention that while vocal abuse does produce the above disease processes, one should look for other than physical reasons in many instances as the cause of such laryngeal pathology. The etiology of these lesions is still obscure—for even on thorough examination we can see a nodule or ulcer without actual contact or overriding. Though strained voice and faulty vocal habits are frequently given as the cause, this is not always the case. In my experience, I have seen contact ulcers and nodules appear rather suddenly during a period of "shock reaction", with subsequent complete disappearance follow-

ing the period of readjustment, without the use of surgical intervention. Particularly in the so-called "contact ulcer", could this not be on a vascular basis through the production of ischemia by vasospasm? The theory of autonomic dysfunction with psychological imbalance does not appear far fetched.

The inadequate individual, may, through over control of the autonomic nervous system, produce the pictures of organic laryngeal pathology. Therefore, I feel that before surgery is offered, it behooves us as physicians to determine if we are faced with a basic neurosis or true vocal abuse. Surgery on the anxious neurotic should be withheld as a last measure, and only when insight has been given. In the majority of these cases the vocal disorder is so interrelated with personality difficulties, environmental problems, and emotional conflicts, that the prognosis at best is poor. We must train ourselves to recognize these personality disorders as related to vocal problems, and as such, weigh the relative importance of functional versus organic pathology. In few of these cases can the laryngologist alone administer adequate therapy. In an attempt to develop a better integrated and more mature personality, psychiatric collaboration is highly advisable. In conjunction with reassurance and vocal exercises by the laryngologist, this will provide the basis for a more favorable prognosis.

CASE REPORTS

Case No. 1.—A 42 year old clergyman with progressive vocal difficulty manifested by pitch fluctuations with varying degrees of hoarseness. On occasion, his vocal efforts would be barely audible. Difficulties became more extreme during the reading of the prayers than on delivery of the sermon. Speech efforts were accompanied by progressive expenditure of actual physical energy with increasingly poorer vocal results. He finally developed a carry-over of speech difficulties into conversation.

The patient was seen by an otolaryngologist who made a diagnosis of "laryngitis" and started a regimen of vocal rest, inhalations, and instillations. This had little effect on the vocal quality, for despite prolonged periods of abstinence from vocalization, his weekly attempts thereat became less audible to his congregation.

When first seen by the author, the patient appeared as an extremely tense and very anxious individual. Marked physical effort on his part was required for recounting his symptoms, during which, the patient was continually drying his hands with his handkerchief. The voice showed fluctuating pitch even in ordinary conversation. A physical examination failed to reveal any positive findings, except for moderate injection of the approximating edges of the vocal cords and some thickening. Because of such a dearth of findings, a diagnosis of functional dysphonia was considered at this time.

Since shortly thereafter, the patient was contemplating a trip which would carry him within the vicinity of one of the nation's leading medical centers, I urged him to seek consultation there. Soon after, a confidental letter from one of our leading colleagues sustained my impression of functional aphonia.

Still seeking another opinion, the patient consulted with another laryngologist, who made the diagnosis of "vocal nodules". For a time, however, this obscured the true picture, for here at last a sound organic diagnosis was suggested.

With consideration for his profession, I delayed any recommendations for other than six weeks of vocal rest. Following this period, with no apparent improvement, I told him that I considered his difficulties functional in nature and probably resulting from some emotional conflict. My recommendation for psychiatric aid was accepted by the patient.

Then followed prolonged psychiatric analysis which revealed a neurotic pattern developed during early childhood as the result of an overprotective and domineering mother who instilled in her son a fanaticism that "he had to be better than anybody". When he realized that he was not measuring up to this standard, the compulsion to achieve marked success made him try harder. The tension kindled into feelings of frustration and conflict. His resentment to the pulpit, and his unconscious hostility to his life as a clergyman was best achieved through vocal difficulty. For, if unable to speak, there was justification for capitulation.

At the present time, as a result of the reassessment of values and personality integration through analysis, and periodic reassurance, the patient's voice has resumed strength and clarity with considerably better control over the pitch.

Case No. 2.—A 51 year old white male with progressive dysphonia which began five months before his visit to my office, had maintained a position as foreman of a large construction firm. With vocal difficulty, he became progressively more reluctant to talk with his workers. He took to alcohol in large quantities. Prior recommendations were direct laryngoscopy with "stripping of the vocal cords".

Examination revealed a markedly tense and anxious individual. Aside from slight thickening

of the vocal cords and some injection of the contacting edges, there was no pathology noted. Every portion of the laryngopharynx and the trachea was visible to mirror examination.

The lack of sufficient findings and the extreme anxiety and tension of the patient made evident the diagnosis of a conversion hysteria based on some emotional conflict. It was my opinion that direct laryngoscopy offered nothing in the diagnosis of this case, and that "stripping of the vocal cords" would have given this patient an organic defense against his conflict. It was therefore recommended that he undergo phychiatric help.

Before accepting this advice, he visited an alert laryngologist in Mississippi, who confirmed my findings, and also felt that any surgical intervention (diagnostic or therapeutic) was contraindicated, and that this patient could only be helped by psychiatric counsel.

Case No. 3.—A 35 year old unmarried white female complained of periodic loss of voice over the past five years. During these years, she saw other otolaryngologists with varying diagnoses of laryngitis, vocal fatigue, nodules, and voice strain.

Her complaints commenced shortly after her recently widowed sister and three children had moved in with the patient, who was already supporting her mother and cardiac invalid father.

Examination revealed a very tense, rather obese female, perspiring freely, and with marked carotid pulsation. There was severe dysphonia. The vocal cords were normal in appearance, but moved very little on respiration. However, with cough, there was no doubt as to the full range of their motility.

The patient was given reassurance, placed on vocal rest, and requested to return within one week. On her second visit, there was still some dysphonia, although considerably better. The tension and anxiety were still apparent, although somewhat ameliorated. Since examination again failed to reveal organic vocal pathology, I decided to tactfully acquaint her with my impression, namely, that this was an hysterical manifestation without organic reason.

The case history was more carefully searched, and my convictions were strengthened that there was a true conflict between her frustrated desires and the knowledge of obligations to her family. Her anger at being placed in such a position, and her inability to express resentment at home, brought about the dysphonia as an unconscious compromise by an increase of attention on the part of her family. The dysphonia was both the expression and way of dealing with the conflict.

Clarification of her position and explanation of the mechanics of her dysphonia, with reassurance, brought about a resumption of normal voice. She has been free of dysphonia for longer than one year.

Case No. 4.—A 56 year old white male employed as postmaster of Class II Postoffice. He was troubled with intermittent dysphonia for approximately one year with periods of exacerbation and improvement. He was a very tense and anxious individual with additional complaints localized to his intestinal tract and frequently accompanied by palpitations. His position as postmaster had proved most trying, since business in the postoffice increased considerably, and the help situation became more critical.

Examination revealed a right vocal cord that was slightly injected on its contact surface, and somewhat thickened compared to the left cord. There was no impairment of motility.

The patient was given reassurance that there were no neoplasia and that the dysphonia was produced through tension and anxiety over his health and his job. He was placed on physical and vocal rest for a period of two weeks, following which he was given a series of vocal exercises to perform at home in addition to encouragement to sing.

He showed steady and sustained improvement in his voice.

I trust that I have made clear the interrelationship between conflict and dysphonia. It is my thought that where unnecessary surgery is performed, a permanent fixation may be created. With emotional conflict as the etiology of these vocal problems, surgery in these cases would render the approach to the true cause much more difficult. Promiscuous and useless surgery has the positive tendency to make the actual conflict less accessible to the patient's awareness since the vocal cord dysfunction was a curtain concealing the conflict.

In conclusion, these are cases which we must be prepared to recognize and help. Such individuals cannot immediately be referred to a psychiatrist, but must be helped in the beginning by the physician who is in a position to prepare the patient to face his problem.

DISCUSSION

Dr. Harold L. Kearney (New Orleans): I agree with Dr. Zurik that there is generally an emotional background in the etiology of both vocal nodules and contact pathology of the larynx. The emotionally disturbed patient almost constantly talks or yells through a glottic spasm and this combination is probably the usual cause of vocal nodules or contact ulcers.

In a paper entitled "Psychophonasthenia" (Transactions of the American Laryngological, Rhinological and Otological Society, 1941) by the late, able and beloved pioneer in the study of speech disturbances Dr. James Sonnett Greene,

there are some remarks in discussion by Dr. J. E. McAskill who says: "The production of speech is a complex, finely balanced muscular activity where many groups of muscles must be perfectly harmonized. Involved are the muscles of breathing, of vocalization, and of articulation. All these groups of muscles must be perfectly harmonized, if one is to have that smooth co-ordination of a well-oiled machine that pours out a streamlined speech. Now, let an emotional turmoil enter the picture,—say,—fear of one's speech,—and something happens at the control. The physiological reaction in the nuclear cells is still a mystery but we do know there is an interruption or a change in the character of the impulses to the end organs, impulses running wild, which throw co-ordination out of gear. The muscles go into a riot. There is speechlessness or one throws out a jumble of mutilated words. The trouble is in the control, not in the vocalizing machine."

My compliments to Dr. Zurik for his able presentation of his subject.

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ARTERIOSCLEROTIC HEARTS AT WORK *

HOWARD HANSEN, M. D.
N. K. WEAVER, M. D.
BATON ROUGE

INTRODUCTION

During recent years there has been increasing awareness by both physicians and laymen that patients with arteriosclerotic heart disease are not necessarily doomed to a state of invalidism, dependency, and early demise. We now realize that in most cases the diagnosis of myocardial infarction or angina is compatible with many years of continued existence, and, under proper circumstances, with many years of productive employment.

Cardiac patients have been gainfully employed at the Esso Refinery in Baton Rouge. During the past three years we have used physical capacities matched with the physical demands of the jobs for proper placement of the partially disabled employees. The method will be described in Part I and the results in Part II of this presentation.

PART I
JOB PLACEMENT OF THE PARTIALLY DISABLED

Development of Present Method

Five years ago there were on record 465 employees with all types of physical disability who could not or would not perform all the physical effort required of their assignments because of actual or alleged physical disability. The employee with arteriosclerotic, phychogenic and/or iatrogenic cardiac disability was difficult, if not impossible, to place in a productive assignment.

The philosophy of achieving proper placement of employees with partial disabilities can be stated briefly:

1. The presence or absence of physical disability is not, per se, of particular significance.

2. Whether or not the physical disability possessed by an employee interferes with the performance of his assigned task is of the greatest significance.

3. If an employee possesses sufficient physical capacities to meet all the physical demands placed on him by his job, full performance is possible despite the presence of disability.

4. The physical capacities (abilities to perform physical acts) of employees and the physical demands placed on employees by jobs, may be evaluated and expressed objectively.

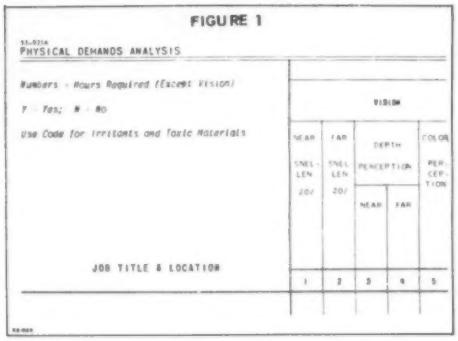
A search of the literature revealed that the method of Hanman [1] could be tailored and modified to suit our needs. In October of 1951, an engineer was assigned the task of gathering data on the physical demands of the jobs in the Refinery. The plant physicians set about to determine the physical capacities of the employees who were not performing the full requirements of their jobs.

The Method

The physical demands (Fig. 1 to 5) in terms of hours per day are matched (Fig. 6.) with the physical capacities of the man. Each form has identical headings and the terminology has the same meaning for laymen and physicians. A booklet describing this method of coping with the problem of occupational handicaps was prepared and discussions were held with

* Presented at the Seventy-fourth Annual Meeting of the Louisiana State Medical Society, May 22, 1954, in New Orleans.

FIGURE 1



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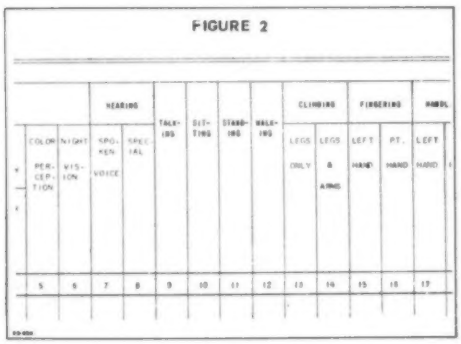
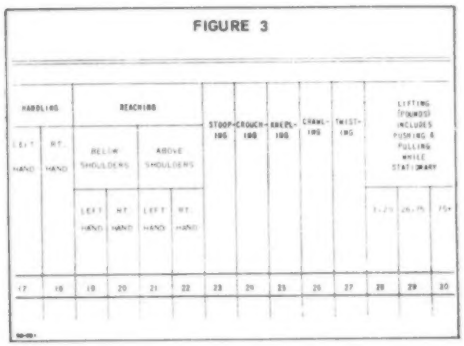


FIGURE 3



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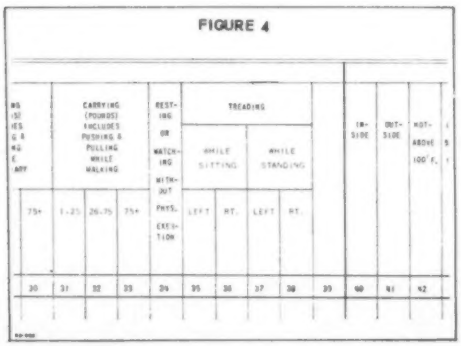


FIGURE 5

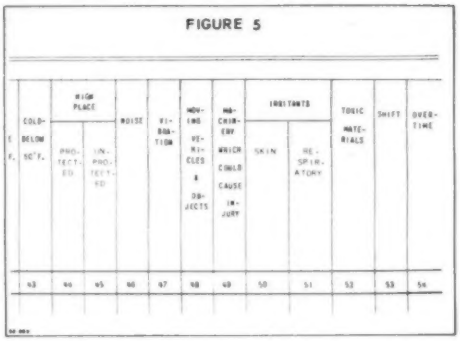


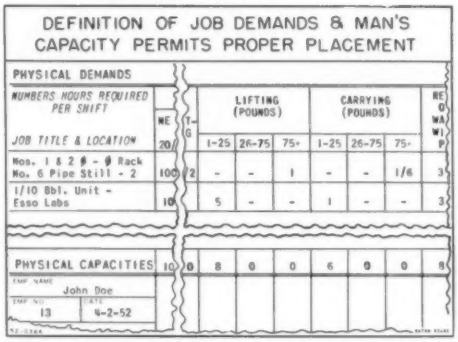
FIGURE 6

DEFINITION OF JOB DEMANDS & MAN'S CAPACITY PERMITS PROPER PLACEMENT

PHYSICAL DEMANDS			LIFTING (POUNDS)			CARRYING (POUNDS)			
NUMBERS HOURS REQUIRED PER SHIFT	NE	T-G	1-25	26-75	75+	1-25	26-75	75+	REQUIRED WAIVED
JOB TITLE & LOCATION	20								
Nos. 1 & 2 ϕ - ϕ Rack No. 6 Pipe Still - 2	100	2	-	-	1	-	-	1/6	3
1/10 Bbl. Unit - Esso Labs	10		5	-	-	1	-	-	3
PHYSICAL CAPACITIES	10	0	8	0	0	6	0	0	0

EMP. NAME: John Doe
EMP. NO.: 13
DATE: 4-2-52

FIGURE 7



all the supervisors and foremen. Proper placement promotes efficiency (Fig. 7). Improper placement is poor business (Fig. 8). New life was given to the handicapped people in our plant because they were at last being assigned to productive work within their capacities.

Discussion

The etiology of an occupational handicap is of no practical value to the foreman, the superintendent, or the employment manager. Frequently, it is a disadvantage because the experience and popular beliefs of the laymen, especially concerning heart disease, are misleading. The negative evaluation of physical capacities, in our experience, has proved equally ineffective because the foreman and others responsible for the employee cannot effectively interpret the capacities by knowing the incapacities. We also believe that the average physician has not been trained in properly evaluating the cardiac patient's residual capacity and he has been overly cautious. He has established unwarranted

FIGURE 7

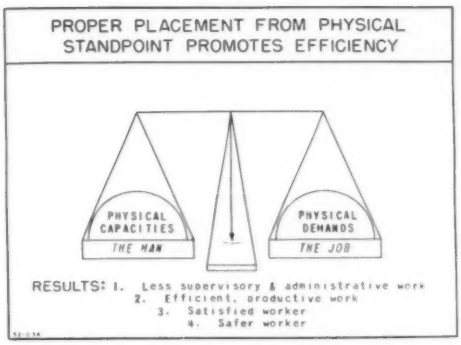
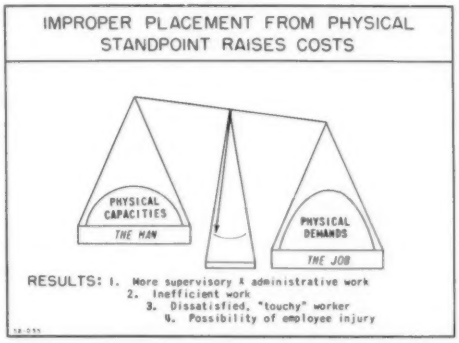


FIGURE 8



fears on the part of employers and his patients.

Matching the physical capacities with the physical demands of the jobs, for the first 265 cases of alleged partial disability for any reason, resulted in only 23 reassignments of work.

The present status of these employees can be summarized as follows:

		Number of Cases	Percent
A.	Died	2	0.8
B.	Totally incapacitated for any assignment	13	4.9
C.	Found to have full physical capacities	89	33.6
D.	Partially disabled but 100% effective in present assignment	99	37.3
E.	Partially disabled—85% to 95% effective in present assignment	16	6.0
F.	Partially disabled—65% to 85% effective in present assignment	27	10.2
G.	Partially disabled—50% to 65% effective	19	7.2
		265	100.0%

The success of our type of "Work Classification Unit" is dependent upon the qualifications of the medical staff to evaluate properly the abilities of the patient and to communicate an understanding of these abilities, with qualifications when indicated, to the management. The evaluating physicians should have training and experience in internal medicine and cardiology and the necessary diagnostic aids to evaluate the total capacities of the patient. The industrial physicians always obtain competent consultations, whenever prudent, to satisfy the patient or his attending physician.

The physical demands of the various types of jobs are not usually objectively evaluated even by a physician in industry. We would like to summarize the maximum, minimum, and average demands for the major exertion factors of the more strenuous laboring types of jobs in our plant:

The maximum amount of walking required of any carpenter in our plant is two and one-half hours per day. The maximum time it would require him to walk up steps or ascend ramps is one hour per day. The maximum time he would be lifting, carrying or pushing and pulling the equivalent of 1 to 25 pounds would be six hours; of 26 to 75 pounds two hours; over 75 pounds, twenty minutes (Fig. 9).

FIGURE 9

PHYSICAL DEMANDS OF CARPENTERS' JOBS

		Hours per Day		
		Max.	Min.	Ave.
Walking		2 1/2	1 1/2	2
Ascending stairs, etc.		1	1/2	3/4
Lifting and Carrying	1 to 25#	6	1	2
	26 - 75#	2	0	1
	Over 75#	1/3	0	1/6

52-058

Usually the exertion is spread over several periods in a day but never exceeds these factors of time and exertion in any eight hour period.

Similar physical demands are shown in Figures 10 and 11 for pipefitters and general laborers.

FIGURE 10

PHYSICAL DEMANDS OF PIPE FITTERS' JOBS				
		Hours per Day		
		Max.	Min.	Ave.
Walking		5	1/2	2
Ascending stairs, etc.		1 1/2	0	1/2
Lifting and Carrying	1 to 25#	6	1	4
	26 - 75#	1	0	1
	Over 75#	3	0	1/6

52-060

FIGURE 11

PHYSICAL DEMANDS OF GENERAL LABORERS' JOBS				
		Hours per Day		
		Max.	Min.	Ave.
Walking		4	1	2
Ascending stairs, etc.		1/2	0	1/5
Lifting and Carrying	1 to 25#	8	0	5
	26 - 75#	3	0	1
	Over 75#	1/2	0	1/10

52-059

In figure 12 we have shown the physical demands necessary to perform 90 and 50 per cent of the manual labor jobs in the plant.

FIGURE 12

CAPACITIES IN HOURS/DAY NEEDED TO DO 90% AND 50% OF ALL MANUAL LABOR JOBS (Skilled & unskilled included)		
	Hours per Day	
	90%	50%
Standing	6	4
Walking	3	1 1/2
Ascending stairs or ramps	1	1/5
Climbing (use of arms & legs)	1	1/5
Lifting up to 75#	2 1/2	1
Carrying up to 75#	1	1/5

52-057

PART II
ANALYSIS OF THE RESULTS OF SELECTIVE PLACEMENT OF EMPLOYEES WITH ARTERIOSCLEROTIC HEART DISEASE

This report is based on an analysis of 82 employees at the Baton Rouge Refinery who are listed in our current disability file as having angina pectoris or healed myocardial infarction, and who have been at work a period of six months or more since the onset of symptoms of cardiac disease. The 50 cases of myocardial infarction all have the characteristic electrocardiographic changes (questionable cases having been discarded) and a history which permits determination of time of onset of the condition. The 32 patients with angina have a typical clinical history of exertional chest pain, and in all cases the diagnosis is supported by ECG abnormalities, which may be apparent only following a Master's exercise test. All of the employees have been placed in a job assignment commensurate with their capacities, where they have worked six months or longer. Our experience indicates that if a patient with arteriosclerotic heart disease survives the acute period of infarction or coronary insufficiency and is able to resume work and be actively employed six months, his prognosis for continued ability to work is good.

Age of Onset

Figure 13 shows the distribution of cases according to age at time of onset of arteriosclerotic heart disease. Seven of the infarcts occurred before the age of forty, the youngest being thirty-two; the oldest was sixty-four. The company's retirement age is sixty-five. In most instances the infarctions or the onset of angina occurred in employees during their fifth and sixth decades, as would be anticipated for an industry with a fairly

FIGURE 13

AGE AT ONSET		
	MYOCARDIAL INFARCTION (50 Cases)	ANGINA PECTORIS (32 Cases)
30 - 39 Years	7	2
40 - 49 Years	15	12
50 - 59 Years	24	16
60 - 64 Years	4	2

57-060

stable employee population.

Years Worked with Arteriosclerotic Heart Disease

Figure 14 indicates the number of years

FIGURE 14

YEARS WORKED SINCE ONSET OF ARTERIOSCLEROTIC HEART DISEASE		
	MYOCARDIAL INFARCTION	ANGINA PECTORIS
1/2 - 2 YRS.	20	8
2 - 5 YRS.	17	16
5+ YRS.	13	8

worked since onset of symptoms for the two conditions, myocardial infarction and angina pectoris. While the average years worked is not long—3.2 for the infarction and 4.0 for the angina patients—it must be remembered the cases were all selected from our current disability file, and the program for selective placement has been in effect only a little over two years. The patients with infarction have worked a total of 163.5 man-years, the patients with angina 112.5 man-years. The combined total for these groups of employees with arteriosclerotic heart disease is 276 man-years of gainful employment.

Types of Work Assignment

Figure 15 shows the distribution of car-

FIGURE 15

WORK CLASSIFICATION		
	MYOCARDIAL INFARCTION	ANGINA PECTORIS
Management	9	0
Technical	7	3
Clerical	4	2
Skilled	19	17
Unskilled	11	10

diac patients among the major job classifications. Actually, in our refinery we have been able to place them in a great variety of assignments — executives, machinists, welders, pipefitters, clerks, process operators, helpers, etc. In all cases the requisite capacities determine the placement of the partially disabled employee rather than some hazy, preconceived ideas regarding "light" and "heavy" occupations. Twenty-two of the patients with healed infarcts were able to return to their regular job with no significant changes, while 28 were placed in assignments with some limitation as regards the more strenuous activities. And for the angina group, after analysis of their capacities, 17 were able to continue in the regular job without modification, while 15 were transferred to other, more limited assignments.

Effectiveness on the Job

It may be rather surprising to learn that approximately 50 per cent of our employees with coronary heart disease were able to continue in a regular assignment. For the others who were transferred into a different job, it must be emphasized that there was no loss in effectiveness or productivity. The partially disabled employee who is properly placed is fully effective. In reviewing the job situations of the 82 patients, only 1 was once classed by management as "substandard"; this individual was a clerical worker whose physical limitations in no way affected his work output.

Symptoms of Cardiovascular Disease

Twenty-eight of the 50 employees with healed infarcts were essentially asymptomatic on the amount of physical activity they followed on and off the job. Ten of them complained of dyspnea, which was classed as mild in 7 and moderately severe in 3. Of the angina group, 9 experienced exertional dyspnea, which was considered mild in all cases. None of the patients had persistent congestive failure, although one of the infarct group had episodes of right and left sided failure which were readily controlled by treatment.

Seventeen of the patients with infarcts experienced typical angina; this was severe in 2; moderately severe in 5 and mild in 10 cases. In the group with angina

without infarction, the symptoms as described were severe in 4 cases, moderately severe in 24 cases, and mild in 5 cases. (The breakdown of angina into severe, moderately severe, and mild categories is based on a study of the frequency of attacks).

Sickness Absenteeism

Only 2 of the 82 employees were found to have an excessive amount of sickness absenteeism from work due to cardiovascular disease. Thus, our experience follows that reported by the American Heart Association and other sources—that patients with arteriosclerotic cardiovascular disease whose condition is stabilized and who are properly placed tend to have lower sickness absenteeism than average.

Figure 16 indicates the number of

FIGURE 16

MONTHS SPENT ON "SICK REPORT" BEFORE RESUMING WORK (50 Patients with 51 Myocardial Infarctions)	
	NUMBER OF PATIENTS
1 Month	12
2 Months	6
3 Months	12
4 Months	8
5 Months	9
6 Months +	4

57-083

months spent on sick report by patients at the time of myocardial infarction. That 12 were off the job a month or less may seem surprising, but this is explained by the fact that some of the cases had minor, localized infarctions with little or no symptoms, and in a few cases the diagnosis was "retrospective". We feel that ordinarily a patient with uncomplicated myocardial infarction can return to "light duty" in about three or four months. Although the patient may feel well and the infarct may clinically "heal" in less time, there are certain practical and theoretical considerations which usually result in delaying the return more than a month or two. Our experience indicates that patients with prolonged invalidism of several months' duration are often unable to resume effective work, due to a disabling cardiac neurosis or symptoms of angina or failure.

Incidence of Cardiac Neurosis

As regards the occurrence of cardiac neurosis—while we recognize that most patients with serious coronary artery disease tend to manifest anxiety over their cardiovascular status, only 6 of the eighty-two employees here reported are considered to have a significant or partially disabling degree of neurosis.

SUMMARY

A method for the placement of partially disabled employees in job assignments, based on matching of the physical capacities of the employee with the physical demands of the job, is presented.

The work and health status of 82 employees with arteriosclerotic heart disease at the Esso Refinery at Baton Rouge are reviewed.

CONCLUSION

"Cardiacs" who are properly placed are effective workers.

REFERENCE

1. Hanman, Bert: Physical Capacities and Job Placement, Nordisk Rotogravyr, Stockholm. (John de Graff, Inc. 1951.)

DIAGNOSIS OF RHEUMATIC FEVER *

JOHN MITCHELL, M. D. †
NEW ORLEANS

The diagnosis of rheumatic fever is a difficult subject to discuss without bringing up the obvious. Since the florid case is not hard to recognize, I shall confine my remarks to those borderline situations where doubt may creep in. Most cases of rheumatic fever have joint pains, murmurs, fever, or laboratory signs of infection, alone or in combination, but it is also painfully obvious that not all joint pains, murmurs, fever, or laboratory changes are caused by that disease. The problem is overdiagnosis or underdiagno-

* Presented at the Seventy-fourth Annual Meeting of the Louisiana State Medical Society, May 21, 1954, in New Orleans.

† From the Department of Pediatrics, Tulane University School of Medicine, and the Charity Hospital of Louisiana at New Orleans.

sis and I shall try to bring out the differentiating points that may be of aid in this situation.

That the problem of rheumatic fever is an important one cannot be denied. At the present time there are at least one million persons in this country who have or have had the disease. Between the ages of five and nineteen years, the incidence varies from 2 to 4 cases per 1000. It is the most common cause of heart disease below the age of forty. The number of adults who turn up with respectable signs of mitral disease in later life without the history of a previously diagnosed or treated rheumatic fever indicate that the incidence may be even higher. It is the major medical crippling disease of childhood and the salvage rate is strongly influenced by early recognition.

As Cassels points out, "Rheumatic fever is a systemic disease which affects several organ systems but does so unevenly and without a constant pattern even in a single system." To construct a pattern in any one case, a large number of variables must be considered. Major manifestations are confined largely to four organ systems: the musculoskeletal, cutaneous, cardiovascular, and central nervous systems.

MUSCULOSKELETAL MANIFESTATIONS

Of most value, and the finding that gives its name to the disease, is the arthralgia. Joint pains are common in children and may be associated with everything from a sore throat to ill fitting shoes. Those due to rheumatic fever are characteristic, for the most part, and can be differentiated from nonrheumatic pains. The latter are seldom seen during the active part of the day but tend to show up just after going to bed. Areas of involvement are usually clearly muscle groups, particularly the calf muscles and tendon areas, most commonly back of the knees. They can be relieved by rubbing, heat, or salicylates and are almost always confined to the lower extremities.

In contrast, the joint pains of rheumatic fever are quite different. They are most conspicuous during the day, beginning in the early morning, although they may also be troublesome at night. They are made worse by activity, heat, or rubbing. Usually they will respond to aspirin, and Cassels makes the categorical statement that arthralgia failing to respond to a blood salicylate level of 30 mg. per cent is not rheumatic in origin. The location is also important. In most cases it is seen in both upper and lower extremities at some point in the disease. Of course, if local heat, tenderness, swelling, or redness is present, the diagnosis is that much easier.

CUTANEOUS MANIFESTATIONS

Skin manifestations can often be of great aid in recognizing this disease. Subcutaneous nodules are considered the most diagnostic, being found only in rheumatic fever and rheumatoid arthritis. Others also can be most suggestive. Nodules, unfortunately, usually appear only during florid, symptomatic rheumatic fever when the confusion is past, but occasionally they may be the only manifestation. Firm, painless, and subcutaneous in location, they are often as easy to see as to palpate. They are most often seen over bony prominences such as the patellae, elbows, scalp, and extensor tendons of hands and feet.

More important than their diagnostic value is their prognostic significance. Gibson interprets their presence to mean usually that the patient has been ill for a considerable time, that he most likely has a severe rheumatic carditis and that his convalescence will be prolonged. Certainly, if they are present the patient's status should be carefully evaluated before he is allowed to be active.

Second only to subcutaneous nodules is the importance of erythema annulare (or erythema marginatum). Many are coming to believe that these lesions have almost the same significance in rheumatic fever as the koplik spot has for measles. Made up of circular or wavy lines of roughly circular pattern, they are often missed unless searched for diligently. Occasionally, the lines are interrupted, giving rise to the lay term, "horseshoe fever". Interest-

ingly, these lesions are not affected by ACTH or cortisone.

Erythema multiforme and erythema nodosum are sometimes seen as complications of streptococcal infection but are not as diagnostic. Erythema nodosum cannot be considered diagnostic of rheumatic fever and when present, cannot be considered to necessarily mean rheumatic activity.

CARDIOVASCULAR MANIFESTATIONS

The gravest sign of rheumatic fever is that of carditis. Here again the situation may not be clear. While the early signs of involvement are alterations in rhythm, rate, and the character of the heart sounds, confusion is most often seen when murmurs are present. Many — perhaps most — children have functional murmurs at one time or another and these are particularly prominent in the presence of fever. Like the nonspecific joint pains, however, these murmurs can be recognized when their characteristics are known. They are usually maximal above the sternum, over the pulmonary area, often in the third and fourth interspaces. They are not blowing in character and have been described as "empty" sounding. Heard best when recumbent, they tend to disappear when erect and during deep inspiration.

The murmurs of rheumatic carditis are characteristic for the most part and are of considerable diagnostic and prognostic value. Earliest appearing is usually a soft, systolic, blowing murmur with apical transmission. It is often variable in intensity. It may signify relative mitral insufficiency from dilatation, actual involvement of the valve or both. Soon after the appearance of this murmur there may develop a diastolic rumble at the apex, sharply localized and in the position of the third heart sound. If the aortic valve is involved, there may be a blowing diastolic murmur beginning with the second heart sound and located in the second and third interspaces to the left of the sternum. Late, of course, the diastolic, presystolic murmur of mitral stenosis appears. Involvement of the pulmonary and tricuspid valves is rare. In addition to the murmurs, tachycardia, dilatation of the heart, and friction rubs are of value.

CENTRAL NERVOUS SYSTEM

Of the major organ system involvements, that of the central nervous system is often the most difficult to recognize early. Any child is apt to be fidgety, many are nervous. Often the process is well advanced before help is sought. Fidgety behavior, too, is quite easy to classify when examined closely. The movements are not continuous and do not repeat themselves. When the child moves purposely the movements disappear. Tics, which are commonly seen in children, are repetitive and stereotyped, always involving the same muscle groups. Facial grimacing, shoulder shrugging, winking, and other such peculiar movements may be seen, repeated in the same manner over and over.

Choreiform movements are distinct from these. They are sudden, aimless and irregular. Muscular weakness may be conspicuous. Writing, drawing, buttoning of the clothes, or anything requiring coordination may be very difficult or impossible to perform. The patient drops things and is frequently scolded for being clumsy. Emotional instability and other psychic manifestations often accompany the disease. Not uncommonly, one side of the body is involved but not the other. The tongue cannot be held quietly when protruded, the forearms are pronated involuntarily when extended above the head and facial expression may change from a broad grimace to a tearful look in a matter of seconds.

Although "pure" chorea probably does exist without rheumatic fever and rheumatic chorea may pursue its whole course without other manifestations, only to show up later, these cases must be considered to have rheumatic fever until proven otherwise and managed accordingly.

OTHER AIDS TO DIAGNOSIS

There are, of course, many other manifestations of less importance in rheumatic fever. In Hansen's series, 10 per

cent had abdominal pain as an isolated finding or during their course. The finding of fever is almost universal but difficult to evaluate. The situation in which an active child has a fever of 99.6° F. in the late afternoon, occasionally reaching 100° F. is a common one. If he happens to have a functional murmur and equivocal laboratory evidences of activity, the diagnosis of rheumatic fever is often suspected. This combination of circumstances is probably the area in which overdiagnosis is most often found. Such temperature curves are not necessarily abnormal and should be viewed with suspicion.

The laboratory does not have much of a specific nature to offer. The sedimentation is at once the most useful and the most misleading of the commonly employed tests. It is a reasonably accurate test for systemic infection or necrosis, nonspecific, and influenced by nonrheumatic tissue changes and serum protein alterations. Rheumatic activity may occasionally go on in the presence of a normal sedimentation rate. Correction with the hematocrit is often misleading in children. Some newer laboratory methods for determining rheumatic activity have been described in the last few years. The C-reactive proteins are probably the most useful of these. They are so-called because of the reactivity of certain abnormal proteins in the serum with the C or somatic polysaccharide of the pneumococcus organism. Their chief advantage lies in their sensitivity and lack of a normal range.

The electrocardiogram may or may not be of aid. Jones and Bland, in the review of the largest series of cases yet reported, found on serial electrocardiograms that only 15 to 20 per cent of children with rheumatic fever showed significant changes during their course.

Lastly, one should have a cautious attitude toward the diagnosis of rheumatic fever. It should never be made unless the situation is reasonably clearcut. It has been said that "in medicine, as in law, the patient should be presumed innocent until proven guilty."

REFERENCES

1. Cassels, D. E.: The diagnosis of rheumatic fever, Pediat. Clin. N. America, (February) 1954.

2. Polk, D. Stewart: Differential diagnosis of rheumatic fever in children without the aid of advanced laboratory facilities, Pennsylvania M. J. 55:917, 1952.

3. McCarty, Maclyn: Present status of diagnostic tests for rheumatic fever, Ann. Int. Med. 37:1027, (Nov.) 1952.

4. Hill, A. G. S.: C-reactive protein in rheumatic fever, Lancet 2:558, (Sept. 20) 1952.

5. Gibson, S.: Eyes, hands, and ears in the diagnosis of heart disease, Pediat. Clin. N. America, (February) 1954.

6. Bland, E. F., and Jones, T. D.: Rheumatic fever and rheumatic heart disease; a twenty year report on 1000 patients followed since childhood, Circulation 4:836, 1951.

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VULVITIS *

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LAFAYETTE

Vulvitis is one of the most exasperating problems in the treatment of women, and it is one that is met with very frequently in general practice. This paper will review some of the leading causes of vulvitis in order to refresh them in our minds.

Simple vulvitis is an acute inflammation of the vulva, associated with pruritis. Its causes are many and varied. In children, gonococcal vulvovaginitis, foreign body in the vagina, irritating urine, lack of cleanliness, trauma, and intestinal parasites may be implicated.[1] The treatment varies with the etiology. The treatment of choice in gonococcal vulvovaginitis is penicillin, or one of the newer antibiotics. In refractory cases, estrogenic suppositories may be used. These, of course, should be inserted by the physician.

In adults, vulvitis may be caused by irritating discharges from the vagina, cervix or uterus. Particularly implicated, are trichomonas, monilia, and mixed vaginitis, as well as discharge from fistulous tracts. A search should be made for allergies,[2] and for contact dermatitis caused by soaps, perfumes, deodorants, powders, and clothing, particularly the synthetic fibers such as nylon. Chemical irritation may occur from douches, contraceptive agents, nail polish, drugs, and laxatives, especially

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those containing phenolphthalein. Occasionally, mechanical irritation due to a sanitary napkin can cause a mild vulvitis. Pediculosis pubis and scabies should be investigated. Sometimes in intractable cases of pruritis vulvae, a psychogenic element can be elicited. Rectal lesions such as fissures and fistulas, as well as intestinal parasites, should be searched for.

The treatment consists of finding the underlying cause and eradicating it, or of desensitizing the patient if an allergen is involved.[2] Wet compresses and a few days' rest with sedatives and Sitz baths will help in the acute stages. After this, soothing ointments and lotions may be used.

Follicular vulvitis, furuncles, and *carbuncles* are localized infections usually due to a staphylococcus. Treatment consists of hot compresses and the newer antibiotic ointments or parenteral penicillin. Occasionally, incision and drainage of a carbuncle may be necessary.

Intertrigo is a chafing and maceration of the skin due to moisture and friction of opposing surfaces. It is frequently due to simple uncleanliness. The condition starts in the folds of the skin and extends outward. Cleanliness of the involved parts is essential in treatment. Soothing ointments and dusting powders such as borated talc will give relief. In obese individuals, reduction in weight is necessary.

Tenia cruris is a fungus infection of the skin caused chiefly by the *Epidermophyton inguinale.* Calamine lotion or fungicidal preparations, particularly the lotions or powders, will effect a cure.

Trichomonas vaginitis is caused by the protozoan *Trichomonas vaginalis.* In this disease, the upper vagina and cervix typically show punctate hemorrhagic spots, giving a "strawberry" or "fleabitten" appearance. The discharge is usually frothy white and profuse. The diagnosis is made by vaginal smear with demonstration of the trichomonas. In the treatment, it is often necessary to cleanse the vagina with green soap. It is then dried with a sponge dipped in ether or mercurochrome. This is usually followed by insufflation with one of the trichomonocidal agents.

Many different preparations have been put on the market for the home treatment of trichomonas vaginitis. Some of these are Floraquin (Searle), Devegan (Winthrop), Virform insert (Ciba), Caprylium (Strasenburth) and Inserferm (Blue Line). Any of these medications can be used, and should be used in conjunction with acid douches. It is essential that treatment be continued through the first menstrual period. In spite of adequate treatment, there will be a certain percentage of failures; these should be studied for foci of reinfection in the bladder, urethra, Skene's and Bartholin's glands, and in the husband's prostatic secretions.[3]

In *Monilia vaginitis* the causative agent is *Candida albicans.* This occurs frequently in pregnancy, and is usually implicated in diabetic vulvovaginitis. The symptoms are pruritis, dyspareunia, and a discharge which is usually not profuse as in trichomonas vaginitis. The vagina often contains a cheesy, white, flaky material. The diagnosis is made by vaginal smear for *C. albicans.*

In diabetic vulvovaginitis it is necessary to control the glycosuria and hyperglycemia before the yeast infection can be alleviated. Ant [4] reports good results with the use of vitamin E suppositories in the treatment of diabetic vulvovaginitis. Moniliasis will usually respond well to painting the vagina with gentian violet, or to the home use of Gentia Gel (Westwood), except in the few patients who are sensitive to the drug. Other useful agents are Propion Gel (Wyeth), Coprylium (Strasenburth) and Inserferm (Blue Line). Again, there will be a certain percentage of failures. These should be studied for foci of reinfection such as the fingernails, etc.

Syphilis—The most frequent location of the chancre is in the posterior fourchette and fossa navicularis.[5] They may also occur anywhere in the vulval area and thighs. Chancres may be erosive, ulcerative, or rarely, papular. They are usually painless. The diagnosis must be made by

darkfield examination for spirochetes. Serologic tests are positive several weeks after the onset of the primary lesion. Secondary lesions are seen more frequently in women, and they take the forms of condylomata lata which are moist, flat papules, and mucous patches. Tertiary lesions of the vulva are rarely seen, but when gumma does occur, it must be differentiated from carcinoma and tuberculosis. The treatment of choice in syphilis is penicillin.

Chancroid is caused by the Ducrey bacillus. The lesions are painful ulcerations that are usually multiple rather than single. The multiplicity is often due to autoinoculations with the formation of "kissing ulcers". There is an irritating discharge, and buboes may occur. The diagnosis is made by smears for Ducrey's bacillus. Sulfa drugs, streptomycin, and aureomycin give good results in the treatment of this disease.

Granuloma inguinale is an extremely unpleasant disease in which a large granulating, ulcerative lesion of the vulva is encountered. This may become very widespread. Secondary infection usually occurs with the formation of a very malodorous discharge. Pseudobuboes may be found. The disease is caused by Donovan bodies which are felt by Anderson et als [6] to be an encapsulated, gram negative, bipolar staining bacterium. The treatment is the use of streptomycin, aureomycin, or chloromycetin.

Lymphogranuloma venereum is of virus etiology. The primary lesion is minor and usually heals within a few days; the patient is generally not seen at this time. Buboes then appear; (the time of their onset varies); and are often accompanied by constitutional symptoms such as fever and malaise. The buboes usually break down to form fistulous tracts. Elephantiasis of the vulva (Esthiomene) may occur as a late complication.[7]

The point that is easiest to remember about this disease is the anogenital syndrome. Proctitis is a common late manifestation, and if there is no treatment, stricture formation may occur. The diagnosis is made by the intradermal Frei test. Sulfa drugs, aureomycin and chloromycetin will cause regression of the lesions and symptoms, but it is questioned whether or not the virus is destroyed by these drugs.

Any of the venereal diseases may occur in combination with the others, and it is impossible to differentiate between them and carcinoma on clinical grounds alone. Conrad Collins [8] found that venereal granulomatous lesions seem to be a predisposing factor in development of vulvar malignancy. Hence, all ulcerative lesions of the vulva should be subjected to the following diagnostic survey: Frei test, smears for Donovan bodies and Ducrey bacilli, serologic tests and darkfield examination for lues, and biopsy.

Lipschutz ulcer is an acute, painful ulceration that is found most frequently in virgins and nulliparas. *The Bacillus crassus* is found on smears, but this is apparently identical with the nonpathogenic Doderlein's bacillus.[5] There is no specific treatment.

Tuberculosis of the vulva is the rarest form of genital tuberculosis. It is usually not primary, but is frequently the result of spread from foci elsewhere in the body. The most frequent type of lesion is the ulcerative one. It usually starts as a submucous or subcutaneous nodule which breaks down to form a chronic, firm ulceration. Lupus-like lesions with their characteristic apple-jelly nodules are next most frequently seen. The lesions are chronic and progressive. The diagnosis is made by biopsy, and the demonstration of the bacilli, if possible. Streptomycin is used in treatment.

Atrophic Vulvitis—Atrophy of the vulva is a normal part of the aging process, and is usually asymptomatic. However, if infection is superimposed on the atrophic change, a chronic atrophic vulvitis may result,[9] with pruritis and occasional dysuria from urine passing over an irritated surface. A search should be made for a causative agent, as outlined in the discus-

sion on simple vulvitis. Estrogens may help, but they are of more value in treatment of senile vaginitis. Bland ointments, sedatives, antihistamines, and vitamins such as A and B may be of help.

Lichenification is a thickened or leathery textured skin resulting from long-standing vulvitis with pruritis and scratching.[9]

Kraurosis Vulvae—The term kraurosis means a shriveling or shrinking up. It is an atrophic change as opposed to the hypertrophic changes in leukoplakia. In this condition, the mucocutaneous surfaces of the vestibule, labia minora, urethral orifice, and clitoris are thin and shriveled with much narrowing of the vaginal orifice. It never involves the labia majora, perineum, or perianal regions.[5] The treatment is essentially the same as in chronic atrophic vulvitis.

Leukoplakia—In this condition, there is the development of thickened areas in the form of parchment-like plaques or irregular patches which are grayish to bluish white in color. Fissures and scratch marks are common. The disease may involve the labia majora, minora, prepuce, perineum, and perianal skin. It is frequently combined with kraurosis. The etiology is unknown, although Miller et al,[10] feel that there may be a chronic irritative urinary factor involved. They found that the urinary organic acids are consistently elevated in this disease.

The diagnosis is made by clinical picture and biopsy. Estrogens are not of much benefit in the treatment of this disease. Vitamin A with hydrochloric acid may help some patients, but it is falling into disrepute.[10] The treatment of choice is still vulvectomy, particularly since leukoplakia is a premalignant disease.

In conclusion, the diseases that affect the vulva are numerous. Most of them will respond to a patient search for the underlying cause, and careful therapy. Some of them, particularly those with an obscure etiology, continue to be a real problem in the practice of Gynecology.

REFERENCES

1. Schauffler, G. C.: Pediatric Gynecology, 2nd Ed., Chicago, Year Book Publishers, 1947.
2. Collins, J. H., and Ellington, C. J., Jr.: Vulvovaginitis, New Orleans M. & S. J., 104:220, 1951.
3. Filler, W.: Gynecology Diseases and Minor Surgery, 1st Ed., Springfield, Illinois, Charles C Thomas Publisher, 1952.
4. Ant, M.: Diabetic vulvovaginitis treated with vitamin E., Am. J. of Ob. & Gyn., 67:407, (Feb.) 1954.
5. Speiser, M. D.: Gynecology Diseases and Minor Surgery, 1st Ed., Springfield, Charles C Thomas Publisher, 1952.
6. Anderson, K., DeMonbreum, W. A., and Goodposture, E. W.: An etiologic consideration of Donovan's granulomatosis cultured from granuloma inguinale in embryonic yolk, J. Exper. Med., 81:25, 1945.
7. Hunt, E.: Diseases Affecting the Vulva, St. Louis, C. V. Mosby Co., 1948.
8. Collins, C. G., Collins, J. H., Nelson, E. W., Smith, C. S., and Mc Collum, E. A.: Malignant tumors involving the vulva, Am. J. of Ob. & Gyn., 62:1198, (Dec.) 1951.
9. Davis, M. E.: Differential Diagnosis and Treatment of Vulvar Lesions, S. Clin. North America, 30:267, (Feb.) 1950.
10. Miller, N. F., Riley, G. M., and Stanley, M.: Leukoplakia of the Vulva, II, Am. J. Ob. & Gyn., 64:768, (Oct.) 1952.

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RETROLENTAL FIBROPLASIA; A BRIEF REVIEW *

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Like the fireball of an H-bomb, retrolental fibroplasia (RLF) has mushroomed in the past decade so that it is now probably the most common cause for blindness in this country and England.[1, 2] Like traffic deaths and household poisonings, increase in frequency of this disease is probably due mainly to new environmental factors, the composition of infants — or the "host factors"—remaining unchanged. While it is most commonly encountered in babies born prematurely,[3 a-e] causal relationships in this association are still far from clear.

Occurring out of any proportion that might be expected simply from better survival rates for these very small infants,[4] this predominantly vascular lesion of the retina must be differentiated from true retinal dysplasias, occurring usually in as-

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sociation with many other serious defects, or from simple persistence of certain embryonic remnants of the hyaloid system.[5, 6] With true RLF, the disorder we are concerned with here, there are no important connotations or associations with congenital defects of any sort; babies with this disease are usually otherwise normal.

Time does not permit a detailed review of ocular embryology as it might be related to RLF, though two factors deserve emphasis:[7-9] The formation of capillaries in the retina is a function of venules rather than arterioles, and capillaries are only formed in veins on the *distal* side of arterioles; this results in a zone free of capillaries adjacent to the arterioles. Only at the periphery of the retina do arterioles and venules join to form arches with which a capillary formation is associated. In stillborns or in premature infants dying early in life, peripheral absence of the internal limiting membrane of the retina, together with a relative deficiency in Mueller's fibers, has also been noted. These peculiarities of the immature retina are undoubtedly important "host factors" bearing on the high incidence of RLF in infants born during a still-transitional phase in development of this structure.[10]

The true incidence of RLF is still unknown and there are many reports indicating wide variations of frequency — from area to area, and among hospitals in the same areas.[14] Much of this variation has been attributed to differences in routine care programs, and much to differences in the frequency and competence of ophthalmologic examinations. Recent published reports place a maximal incidence in infants with birth-weights under 3 pounds as high as 83.3 per cent, though most authors describe typical lesions in a smaller proportion—usually from 20 to 50 per cent of their very small babies.[15-17]

Though typical funduscopic features[18] may be noted in the first few days of life, these are most frequently first seen at about the time when the infant would have attained gestational maturity;[19] i.e., if the infant is born about six weeks prematurely, then retinal changes might be expected to appear when the infant is about six weeks old.[14]

It is encouraging to know that there is a striking tendency for spontaneous regression and healing from any of the early stages of RLF,[20, 21] and just recently there have been noted rather remarkable spontaneous improvements in advanced lesions formerly considered to justify a hopeless prognosis for any useful vision. It is now believed that about half to three-fourths of those patients who develop early lesions will show regression.

While many suspected causative or contributory factors have been studied, major interest at present centers about the effects of oxygen given in high concentrations to premature infants early in life.[22-31] The very efficiency of modern incubators for easily (too easily?) maintaining high concentrations of oxygen may well be considered a very important environmental factor influencing the remarkable increases in RLF during the postwar years. Our Premature Unit at Charity Hospital has furnished many observations on this point to a carefully controlled nationwide study, being conducted under the auspices of the Kresge Eye Institute. While the results of this large investigation will not be ready or available to us until next October—when they will be released simultaneously to the American Academy of Pediatrics and to the American Academy of Ophthalmology—it is already apparent at this early date that *survival* of very small premature infants cannot be related to high or low concentrations of oxygen employed in this study.

All clinical investigations as to etiology or therapy of this lesion are difficult to evaluate because of the remarkable tendency to spontaneous remission previously noted. In early reports, a number of proprietary water-miscible dietary supplements were incriminated, only to be cleared by later studies.[32] The role of vitamin E in prophylaxis or therapy has followed a similar pattern[33, 34] so that at

present its relation to RLF appears to be negligible, or at best merely coincidental. Early exposure to or protection from light has been investigated also, but no relationship has been established.[14]

Studies are continuing on the effects of electrolyte contents of various formulae and parenteral fluids; while Hepner and Krause[21] found a clinical correlation between the incidence of RLF and high intakes of electrolytes, and were able to produce ocular lesions in young kittens given *large* excesses of water, electrolytes, and blood, the practical relationship of their observations to the clinical disease in humans is not yet clear. Ingalls found an epidemiologic relationship to fetal anoxia from various causes,[34, 10] and Szewczyk noted that retinal changes appeared shortly after premature infants were removed from high oxygen atmospheres.[22] From this observation he evolved a theory of relative hypoxia as an important causative factor and proposed the term "hypoxic retinopathy" for the early stages of RLF. Campbell proposed a direct toxic effect of oxygen on immature retinal vessels following her study of the disease in a large premature unit before and after oxygen was routinely employed in early care. She related the increasing incidence to high concentrations of oxygen given over long periods of time.[24]

Thus, as to the effects of oxygen, there appear to be several important considerations—relative hypoxia from sudden reduction of oxygen concentration, direct toxic effects of high concentrations, and of course the underlying causes for which oxygen was employed in the first place!

In addition, we have also proposed that variations in the impurities contained in supplies of hospital oxygen at different times and places might explain the variations in the incidence and severity of lesions noted from season to season, area to area, and among hospitals in the same area; studies are in progress here and elsewhere at present to explore this possibility further.

Since Patz and his co-workers,[27, 30] and also Ashton and his group[31] have developed reasonable technics for producing the disease in experimental animals, it may be possible to clarify further these apparently confusing or contradictory observations and conjectures. We have great hopes that observations from such research, properly correlated with the ongoing cooperative national study of this disease in many medical centers, may answer a number of questions which now plague all of us interested in the care of premature infants.

At present, and from a very practical point of view, the following statements seem justified by our present knowledge:

1. In nurseries where the incidence of RLF is low, changes in care program should be made only with great caution and only after careful consideration of possible causal relationships to RLF.

2. The fundi of all premature infants should be repeatedly and carefully examined, during hospitalization and for several months (three to six months) after discharge.

3. For the present, at least, oxygen should be employed sparingly, and only for specific and highly individualized indications.

4. When oxygen must be used for valid reasons, its concentration should be carefully measured and adjusted by trained persons.

5. All efforts should be made to avoid or promptly treat all disturbances in fluid or electrolyte balance, by meticulous attention to isolated technics, proper use of prophylactic agents for controlling infections, careful regulation of intakes related to individualized needs, and by early recognition with prompt therapy of any disturbances which may occur despite such precautions.

6. Like many diseases which might be significantly related to purely environmental considerations, or which will require planning for long-term care, RLF should be reportable.

Addendum

Since this manuscript was prepared, a

review of experiences with this problem at Bellevue Hospital in New York City has been published,[36] with the conclusion: "We believe that RLF is directly related to the excessive administration of oxygen and can be controlled by severely limiting oxygen therapy to premature infants".

REFERENCES

1. Reese, A. B. and Blodi, F. C.: Symposium on retrolental fibroplasia, New York State J. Med. 52:2869, 1952.

2. Bembridge, B. A., Coxon, M., Houlton, A. C. L., Jackson, C. R. S. and Smallpeice, F.: Retrolental fibroplasia: A problem of prematurity, Brit. M. J. 4760:675 (March) 1952.

3a. Terry, T. L.: Extreme prematurity and fibroplastic overgrowth of persistent vascular sheath behind the crystalline lens, Am. J. Ophth. 25:203 (Feb.) 1952.

3b. Terry, T. L.: II. Report of cases, A.M.A. Arch. Ophth. 29:36 (Jan.) 1943.

3c. Terry, T. L.: III. Embryological studies, Am. J. Ophth. 25:1409 (Dec.) 1942.

3d. Terry, T. L.: IV. Etiologic factors, Arch. Ophth. 29:54 (Jan.) 1943.

3e. Terry, T. L.: V. Further studies on fibroplastic overgrowth of persistent tunica vasculosa lentis, Arch. Ophth. 33:203 (Mar.) 1945.

4. Kinsey, V. E. and Zacharias, L.: Retrolental fibroplasia: Incidence with different treatment given infants, etc., J.A.M.A. 139:572, 1949.

5. Reese, A. B.: Persistence and hyperplasia of primary vitreous: Retrolental fibroplasia—two entities, Arch. Ophth. 41:527 (May) 1949.

6. Reese, A. B. and Blodi, F. C.: Retinal dysplasia, Am. J. Ophth. 33:23, 1950.

7. Duke-Elder: Textbook of Ophthalmology, volume 1, pp. 350-357.

8. Michaelson, I. C.: Mode of development of the vascular system of the retina, with some observations on its significance for certain retinal diseases, Tr. Ophth. Soc. U. Kingdom, 68:137, 1948.

9. Mann, Ida: Developmental Abnormalities of the Eye, Cambridge Univ. Press, pp. 206-208, 1937.

10. Ingalls, T. H. and Purshottam, N.: Oxygenation and retrolental fibroplasia, New England J. Med. 250:921 (Apr. 15) 1954.

11. Reese, A. B., Blodi, F. C., and Locke, J. C.: The pathology of early retrolental fibroplasia with analysis of eyes of newborn and stillborn infants, Am. J. Ophth. 25:1407 (Oct.) 1952.

12. Friedenwald, J. S., Owens, W. C. and Owens, E. U.: Retrolental fibroplasia in premature infants. III. The pathology of the disease, Tr. Am. Ophth. Soc. 49:207, 1952.

13. Heath, P.: Pathology of retinopathy of prematurity: Retrolental fibroplasia, Am. J. Ophth. 34:1249 (Sept.) 1951.

14. M & R Laboratories Pediatric Research Conference on Retrolental Fibroplasia, April 28th, 1951.

15. Zacharias, L.: Retrolental fibroplasia: A survey, Am. J. Ophth. 35:1426.

16. Moffat, P. M.: Clinical aspects of retrolental fibroplasia, Proc. Roy. Soc. Med. 43:223 (Mar.) 1950.

17. Guy, L. P., Dancis, J. and Lanman, J. T.: Retrolental fibroplasia, Am. J. Ophth. 36:85 (Jan.) 1953.

18. Szewczyk, Thaddeus S.: A Classification of Retrolental fibroplasia, Am. J. Ophth. 36:1333 (Oct.) 1953.

19. Apple, C.: Retrolental Fibroplasia, Am. J. Ophth. 37:68 (Jan.) 1954.

20. Bedrossian, R. H., Carmichael, P. and Ritter, J.: Retinopathy of prematurity and oxygen. Part I. Clinical study. Part II. Further observations on the disease. Am. J. Ophth. 37:78 (Jan.) 1954.

21. Hepner, W. R. and Krause, A. C.: Retrolental fibroplasia: Clinical observations, Pediat. 10:433, 1952.

22. Szewczyk, T. S.: Retrolental fibroplasia: Etiology and prophylaxis, Am. J. Ophth. 34:1649, 1951.

23. Campbell, F. W.: Influence of a low atmospheric pressure on the development of the retinal vessels in the rat, Tr. Ophth. Soc. U. Kingdom 71:287, 1951.

24. Campbell, K.: Intensive oxygen therapy as a possible cause of retrolental fibroplasia, Med. J. Australia 2:2 48-50 (July 14) 1951.

25. Bousquet, F. P., Jr. and Laupus, W. E.: Studies on the pathogenesis of retrolental fibroplasia, Am. J. Ophth. 35:64, 1952.

26. Ingalls, J. H., Tedeschi, C. G., and Helpern, M. M.: Congenital malformations of the eye induced in mice by maternal anoxia, with particular reference to the problem of retrolental fibroplasia in man, Am. J. Ophth. 35:311, 1952.

27. Patz, A., Hoeck, L. E. and de la Cruz, E.: Oxygen administration in retrolental fibroplasia, Am. J. Ophth. 35:1248, 1952.

28. Crosse, V. M. and Evans, P. J.: Prevention of retrolental fibroplasia, A.M.A. Arch. Ophth. 48:33, 1952.

29. Locke, J. C. and Reese, A. B.: Negative role of light, mydriatics and ophthalmoscopic examination in retrolental fibroplasia, A.M.A. Arch. Ophth. 48:44 (July) 1952.

30. Patz, A., Eastham, A., Higginbotham, H. and Kleh, T.: Oxygen studies in retrolental fibroplasia. II. The production of the microscopic changes of RLF in experimental animals, Am. J. Ophth. 36:1511, 1953.

31. Ashton, N., Ward, B. and Serpell, G.: Role of oxygen in genesis of retrolental fibroplasia, Brit. J. Ophth. 37:513 (Sept.) 1953.

32. Kinsey, V. E. and Chisolm J. F.: Evaluation of several changes in dietary supplements of prematures with respect to the incidence of retrolental fibroplasia, Am. J. Ophth. 34:1259 (Sept.) 1951.

33. Owens, W. C. and Owens, E. U.: RLF in premature infants, Trans. Am. Acad. of O. & O., pp. 18-41 (Sept.-Oct.) 1948.

34. Owens, W. C. and Owens, E. U.: Retrolental fibroplasia in premature infants. II. Studies on the prophylaxis of the disease. The use of alpha-tocopherol acetate. Am. J. Ophth. 32:1631 (Dec.) 1949.

35. Ingalls, T. H.: Congenital encephalo-ophthalmic dysplasia: Epidemiologic implications, Pediat. 1:3, 315-325 (March) 1948.

36 Lanman, J. T., Guy, L. P., and Dancis, V.: Retrolental fibroplasia and oxygen therapy, J.A.M.A. 155:223, (May 15) 1954.

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RETROLENTAL FIBROPLASIA *

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Prior to 1942, it was unusual for ophthalmologists to be called in consultation to see newborn babies; now we frequently see babies, especially prematures, for eye examination. This rather startling change

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came about after Terry [1] published a brief report on a new disease of premature infants which he called retrolental fibroplasia (RLF). He noted that it was a disease in which an abnormal membrane formed behind the lens after birth. He also observed that there were no hereditary factors, and that some new factor had arisen to cause the condition. The identity of this new factor has not been proven, but it seems to be near a solution.

Through the past twelve years the reports on RLF have been voluminous. Most of them have been based on clinical observation, some have been reports on autopsy findings, and recently there have been several reports on the disease experimentally produced in animals. Through these many reports the sequence of changes in the RLF picture has been established.

I particularly want to bring to your attention the recently published classification of the disease by Reese, King, and Owens,[2] who served as a committee appointed for that purpose by the National Society for the Prevention of Blindness. This classification greatly simplifies for the ophthalmologist and the pediatrician any discussion of the disease. Since the classification contains a good description of the ophthalmoscopic findings, it is here quoted in full.

The classification groups the disease into its two phases, the active and the cicatricial phases, and each phase is subdivided into five stages:

STAGES OF RETROLENTAL FIBROPLASIA IN THE ACTIVE PHASE

I. Dilatation and tortuosity of retinal vessels
   Hemorrhages may or may not be present
   Early neovascularization especially in the extreme periphery of the visible fundus may be present

II. Stage I. plus neovascularization and some peripheral clouding
   Hemorrhages are usually present
   Vitreous clouding may or may not be present
   Spontaneous regression may occur

III. Stage II. plus retinal detachment in the periphery of the fundus
   Spontaneous regression unlikely

IV. Hemispheric or circumferential retinal detachment
   Elevation of the retina over a large area, but still with some retina in position

V. Complete retinal detachment

GRADES OF RETROLENTAL FIBROPLASIA IN THE CICATRICIAL PHASE

I. Small mass of opaque tissue in periphery of the fundus without visible retinal detachment
   The fundus may have a pale appearance
   The blood vessels may be attenuated

II. Larger mass of opaque tissue in periphery of the fundus with some localized retinal detachment
   The disc is distorted by traction toward the side of the tissue, which is usually temporally
   Cases ending in Grade I. or II. have useful vision

III. Larger mass of opaque tissue in periphery incorporating a retinal fold which extends to the disc
   Visual acuity varies from 5/200 to 20/50

IV. Retrolental tissue covering part of pupillary area
   Small area of attached retina may still be visible or only a red reflex over a sector of the fundus may be seen

V. Retrolental tissue covering entire pupillary area
   No fundus reflex present

There will probably be some objection to this classification inasmuch as Szewczyk [3] has noted the presence of peripheral retinal clouding or edema antedating any visible changes in the retinal vessels. Guy [4] has noted as the earliest sign a vitreous haziness and myopia. However, even though there may be minor differences in the exact sequence of changes, the grouping will be of great

help in description of findings in the disease.

The matter of giving a prognosis in RLF has been difficult. It has always been necessary for the ophthalmologist to be guarded in giving a prognosis, for it has been no easy matter to determine to his own satisfaction which child might have useful vision, very defective vision or no vision. The classification quoted gives us some help in this problem. The changes which can be completely reversible are differentiated from those which will result in a permanent visual defect. However, here again, one must be cautious in giving a prognosis. For example, a child under our observation, now aged 6 years, was first seen at the age of 6 months. At that time there was retrolental tissue covering part of the pupillary area and there was a red reflex over a sector of the fundus. This was the typical Grade IV. of the cicatricial phase. The other eye at 6 months appeared normal. Now we can see in the extreme periphery of the fundus several small atrophic pigmented areas, but no membrane formation in any part of the eye. The pigment distribution throughout the fundus is not much different than that seen in many myopic eyes of minus 6.00 diopters refraction. However, the best corrected vision is only 20/50. This was probably a Grade I. or Grade II. while in the active phase during the first weeks after birth; spontaneous regression occurred but completely normal vision did not develop.

Similar subnormal vision has been found in several children we have seen who are now old enough for accurate visual acuity tests. One cannot forecast with complete accuracy the ultimate vision in even the mildest forms of the disease, and in making a prognosis one must remember that the visual outcome will probably be worse than the fundus picture would indicate.

Surgical treatment in the cicatricial phase with membrane formation was attempted during the early days of retrolental fibroplasia but no procedure was successful. Several forms of medical treatment attempted were also completely unsuccessful. The most recent reports [3, 5, 6] tend to incriminate abnormal oxygen concentrations and may lead to the answer. The solution of the problem lies entirely in prevention, and we hope this disease which appeared so suddenly twelve years ago may soon leave the scene with equal abruptness.

REFERENCES

1. Terry, T. L.: Extreme prematurity and fibroplastic overgrowth of persistent vascular sheath behind each crystalline lens. Amer. J. Ophth. 25:203, 1942.

2. Reese, A. B., King, M. J., and Owens, W. C.: A classification of retrolental fibroplasia, Amer. J. Ophth. 36:1333, 1953.

3. Szewczyk, T. S.: Retrolental fibroplasia and related ocular diseases, Amer. J. Opth. 36:1336, 1953.

4. Guy, L. P., Dancis, J., and Lanman, J. T.: Retrolental fibroplasia: Ophthalmoscopic findings during the first two weeks of life, Amer. J. Ophth. 36:85, 1953.

5. Patz, A., Eastham, A., Higginbotham, M. S., and Kleh T.: Oxygen studies in retrolental fibroplasia, Amer. J. Ophth. 36:1511, 1953.

6. Lanman, J. T., Guy, L. P., and Dancis, J.: Retrolental fibroplasia and oxygen therapy, J.A.M.A. 155:223 (May) 1954.

The Journal of the Louisiana State Medical Society

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INVESTIGATION OF TAX EXEMPT FOUNDATIONS

"The power to tax is the power to destroy." This statement was made in a Court decision generations ago. Its full significance is not appreciated by the voting public. A corollary may be phrased to read: "Freedom from taxation may result in progressive increase in power."

The importance of such thoughts as these to the physician is that tax exempt foundations may influence government, and have already had much influence in the field of science in its applications to the broad aspects of medicine. Widespread concern as to tax exempt foundations has developed recently. A Congressional inquiry was authorized. A special committee to investigate tax exempt foundations rendered a report on December 19, 1954. The majority report was signed by three Republicans on the five man committee, Chairman Reece (Tennessee) and Representatives Wolcott (Michigan) and Goodwin (Massachusetts). Goodwin, however, signed with strong reservations and dissent from many of its findings and conclusions. The two Democrats on the committee, Representatives Wayne L. Hays (Ohio) and Mrs. Pfost (Idaho) are alleged to have called the report a "crackpot view by persons ill with fear sickness." The sessions of the committee were not harmonious. On August 20, Congressman B. Carroll Reece, Chairman, made a speech on the floor of the House of Representatives, in which he commented on the sessions and stated, "Throughout the hearings Mr. Hays, (Wayne L. Hays of Ohio), assumed an attitude of aggressive suspicion and insulting distrust of the majority members of the committee and of the committee staff." In this speech it was further stated that there were presently some seven thousand tax exempt foundations with assets in excess of ten billion, and with an annual income in excess of three hundred million. These tremendous resources are taken largely out of our taxable income. He further stated that the committee, "had been subjected to various and strange pressures and harrassments. It began to be reviled from many directions very early in its career," and that it had been attacked by several of the major newspapers.

The committee report which was made four months following this speech made the startling charge that the Nation's biggest tax free foundations were acting as a powerful interlocking "intellectual cartel," which it said is promoting a socialism of far greater menace than communism. The report dealt mainly with large foundations in the social science

fields. It mentioned most often the three big foundations, the Ford Foundation, the Rockefeller Foundation, and the Carnegie organizations and their offshoot groups. The train of thought was that the foundations are growing rapidly for tax reasons. Because of their special tax status they are, in effect, spending public money. They exert through the purse "enormous power" in molding the country's thinking. It was not charged that this came into being as the result of an over-all conscious plan, but that the effect of their collective influence was important. Among suggestions made by the Reece report was that more complete financial reports from the foundations should be made public, and that a time limit of ten to twenty-five years on the foundations should be established.

In opposition to the statements made in the report of the House Committee, spokesmen for various foundations stated that the accusations were false; that the investigation of foundations was biased from start to finish; or that flimsy allegations have been accepted as fact without support of trustworthy evidence. Dean Rusk, President of the Rockefeller Foundation, is reported as concluding:

"In addition to many ill-considered recommendations, the Reece committee's report includes two recommendations which the Rockefeller Foundation has strongly supported, namely, full publicity for foundation activities and an increase in the manpower of the internal revenue service to enable it to watch these activities more closely." At this point, members of the medical profession wonder what its attitude should be. The situation is not unlike that which develops in many medicolegal cases where opposing teams of experts take the same facts and draw diametrically opposite conclusions. We recognize the great service that some of the foundations have rendered to the science of medicine in the years, and occasionally, to the practice of medicine. We also recognize the many limitations on the capacity of government to spend money wisely. The foundation money is community surplus, which we hope is being spent wisely. On the other hand, the charge that some foundations are promoting Fabian socialism is disturbing. Since the day of the first income tax law we have already come that road too fast and too far. The advice of Dean Rusk that the public have access to the knowledge of the activities of these foundations is reassuring. And we are reasonably certain that if their activities were patterned along the lines of the American Medical Education Foundation, which is to prevent the advance of socialism in medical schools, the sources for criticisms would be minimized.

It should be quite clear, therefore, from these various points of controversy that the public, as well as the medical profession, has a proper interest in the activities of tax exempt foundations.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1955 ANNUAL MEETING

COMMITTEES ON ARRANGEMENTS

Below are listed the Advisory Committee and chairmen of special committees incident to arrangements for the 1955 Annual Meeting who have been appointed by the General Chairman, Dr. J. Theo Brierre. These doctors should be contacted for information pertaining to various phases of the meeting.

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WHAT THE AMA DOES FOR ITS MEMBERS

C. GRENES COLE, M. D.

We would very much like to see all the doctors of this State, and especially all members of the Louisiana State Medical Society, visit the A.M.A. headquarters at 535 North Dearborn Street, Chicago, housed in a nine-floor building, completely furnished with all the latest equipment so necessary for their 900 employees. These employees carry on, year by year, the many activities and duties required by them in the respective fields to which they are assigned, in achieving the goal to which the National Association is pledged: "the promotion of the Science and Art of Medicine and the betterment of health conditions throughout the nation". Such a visit would open the eyes of some of our more skeptic members who are always talking about how little they get out of the $25.00 annual membership dues to the AMA. You can receive plenty for your dues if you choose and would only take advantage of the many services offered you.

We wish to outline briefly a few of the major services provided you for the $25.00 charged for your annual membership dues.

To begin with, the major portion, $15.00, of your dues goes for a year's subscription to the Journal of the AMA, one of the most recognized and outstanding publications in the world, going to over 160,000 doctors each week. If you prefer a specialty journal you may substitute any one of the following nine specialty journals published by the Association: the American Journal of Diseases of Children, Archives of Neurology and Psychiatry, Archives of Dermatology and Syphilology, Archives of Surgery, Archives of Ophthalmology, Archives of Otolaryngology, Archives of Pathology, Archives of Internal Medicine, and Archives of Industrial Hygiene and Occupational Medicine.

The reading of your weekly Journal of the AMA, one of the best recognized and most extensively read scientific medical publications in the world, will keep you posted as to what medical science is offering in the way of advancement in all fields of endeavor. It will furnish you with the most accurate and trustworthy information obtainable in all fields of scientific research and covers all branches of medicine.

In the past, for years, the physicians had to rely almost entirely on their personal experience in the use of drugs for their patients. Now, you have accessibility to the wonderful service offered you by the AMA's Council on Pharmacy and Chemistry which regularly reports on the therapeutic value of the newer drugs placed on the market, and publishes the result of their examination in the Journal. You, as a member, can also write the Council on Pharmacy and Chemistry, the Council on Foods and Nutrition or the Council on Physical Medicine and Rehabilitation or any of the other Councils for any information desired on certain drugs, foods or other products, or any information you might wish on other subjects so vital to you in the practice of medicine.

AMA does many other things for you, assures you of a good medical education and has been helping you since you first entered medical school. Since 1847, AMA has exerted its very best efforts to improve our medical schools. From 1905 to 1920 it led the fight to eliminate all low rate medical schools and maintain, for those few schools which withstood the acid test of rigid inspection and earned for themselves the designation of Grade-A medical schools, the high standard of education enjoyed at the present time and offered prospective students who are entering our medical schools today. Of course, your Association is continuing to work for better doctors of the future, and is demanding higher standards and better conditions, for our medical schools.

In 1950, the Association organized the American Medical Education Foundation for the purpose of raising funds to help support our medical schools, so desperately in need of money, to continue their high educational standard. Your Association has been a great source of financial aid to these schools, having contributed most generously to this Foundation. Its gift the first year was $500,000 and since then large amounts have been and are being contributed.

AMA secures for our interns and residents, better hospital facilities for their training by repeated inspections of these institutions, and only hospitals having sufficient patients, adequate equipment and competent teaching staffs are approved as teaching institutions for intern and resident training.

AMA maintains a national placement service to aid you in getting properly and happily located after graduation. This service keeps files on communities in need of physicians and physicians in search of locations, and works closely in cooperation with the State Societies in this work. The names of all physicians about to be released from military service are given to the AMA by

the government and through the efforts of AMA and State placement services, these doctors are able to contact communities where they might locate for practice.

Your Association's library, which receives about 2000 leading medical journals of the world, is maintained entirely to be of service to its members. This service is free and permits a member to borrow copies of any of these journals, checking out up to three at one time and keeping them for five days.

A popular service, known as the "Package Library", is carried for the benefit of members. This covers articles from current medical journals, pamphlets and numerous paper-bound publications which are daily being filed and cross-indexed in the AMA library. If you wish such a loan package to review any subject or to prepare a paper, just write the AMA Library, 535 N. Dearborn Street, Chicago, Illinois, and the staff will mail you the information free of charge. As a rule, this service requires two weeks. However, if there is need for quick service, so specify in your request and the material will then be furnished you quickly by airmail, special delivery. In this instance you will be expected to pay the extra postage. You should state clearly and specifically what you want, to permit the staff to select the most helpful articles from the large files.

As a service to the members, a Cumulative Index Medicus is published quarterly, at a loss, listing articles appearing in leading medical periodicals. A separate section is dedicated to books published on medical subjects.

Your AMA makes available to the members, two national scientific programs each year. The Annual Session, which twelve to twenty thousand physicians attend, is held in June. This meeting has been described as "a complete post-graduate education under one roof". The Clinical meeting, with an attendance of three to five thousand M. D's meets each year in early December. This Session features clinical demonstrations, question and answer forums, motion pictures, color television and great numbers of scientific and technical exhibits of profound interest to the profession.

Your Association passes on the merits of medical devices connected with diagnosis and therapy, foods used for special diets, and cosmetics. Members of the appropriate Council study and carefully consider these products and the data relating to them, prior to their acceptance or rejection by the AMA. Reports on the qualities or merits of such products, are published in the Journal under the headings of the Council on Physical Medicine and Rehabilitation, the Council on Foods and Nutrition, and the Committee on Cosmetics. Reports of the Council on Physical Medicine are published in a pamphlet called "Apparatus Accepted", a copy of which will be furnished you free on request. The Council on Foods and the Council on Physical Medicine supervise the publication of the "Handbook on Nutrition" and the "Handbook of Physical Medicine and Rehabilitation".

Any questions regarding drugs or other products related to the practice of medicine will be freely answered by your Association. Thousands of letters from members, containing pertinent questions, are answered annually. Only those medical products which are Council accepted can be advertised in the AMA Journal or the nine AMA specialty journals, or exhibited at AMA meetings, and this serves as a guide to good products.

Your dues,—the ten dollars left after the fifteen dollars is deducted for a year's subscription to the Journal, help pay for AMA's evaluation of drugs and other medical products, a service provided entirely for the benefit of our members and with no charge or obligation to manufacturers. The Seal of Acceptance cannot be bought and only quality products receive this approval. Due to the fact that these seals are so greatly desired by the manufacturers this evaluation program has been extremely valuable to the members in making available to them only quality products.

Special reports for publication in the AMA Journal are prepared by the Scientific Councils on such subjects as Industrial Medicine; National Emergency Medical Service; Nutrition, relation of soil fertility to the nutritional quality of food; and Pesticides. The Committee on Mental Health will also issue special reports. Reprints, of the above mentioned reports are, as a rule, available to members.

Space and time will not permit any detailed statement of other services your AMA offers to members on numerous other subjects and projects. We will only mention briefly these additional services. Your AMA assists research on therapeutic problems financially and by cooperation with clinics and pharmaceutical firms. Recently, in a study of the use of steroids in cancer of the breast, forty-five clinics and fourteen pharmaceutical firms cooperated. Five hundred dollars to individual investigators was authorized and an average of thirty-five to fifty such grants are made annually.

Reports are also made on toxicity of new drugs. Information on quacks and nostrums is available by writing to the Association's Bureau of Investigation.

The Association helps assure good hospitals for you, as a member of the Joint Commission on the Accreditation of Hospitals and also lends financial aid to the Commission.

Your medical records in hospitals are filed by AMA Classification, and in 1937 the Association

edited "Standard Nomenclature of Diseases and Operations", used by seventy percent of hospitals in the United States today.

Your assistants are better trained because of AMA's inspection, classification and approval of schools for laboratory technicians, occupational therapists, physical therapists, X-ray technicians.

AMA offers Public Relations tips for your secretary or nurse in the manual, "Winning Ways with Patients", a copy of which will be sent you on request. Public Relations aids for doctors are available, and a pamphlet on office public relations for the practicing physician will be mailed to you free of charge upon request to the AMA.

You can help in filling that waiting room table by requesting from AMA copies of many attractive pamphlets for this purpose. Some of these would include "Your Money's Worth", "A Doctor for You", a cartoon-style pamphlet, "The Amazing Story". Reprints of informative magazine articles may be obtained. You may, also, secure public relations literature free by writing to your Association's Public Relations Department.

By writing to AMA's Bureau of Health Education, you may secure pamphlets including, "How to be a Good Patient", "Getting Ready for Married Life", "Adoption", "Master Key to Restful Slumber", to reinforce your verbal instructions to your patients. Single copy free,—quantities will be furnished you at cost of publication.

"Today's Health", published by AMA will teach your patients about sound health habits and preventive medicine and should be a "must" in your waiting room. It is offered to physicians at a special low rate of $1.50 per year.

Information on medico-legal problems can be obtained by you or your attorney on a loan basis. In addition to this service, the attorneys on the staff of AMA's Bureau of Legal Medicine and Legislation will gladly answer inquiries on medico-legal problems pertaining to partnerships, selling a practice, relationships between injury and specified disease, ownership of X-rays, income deductions and medical testimony in general. "Abstracts of Court Decisions on Medico-legal Cases" may be purchased for permanent reference from the "Order Department" at AMA headquarters.

AMA represents you before Congress. This is a most important and valued service of your Association, studying all bills before Congress and supporting or opposing said bills as they may affect organized medicine. This service necessitates the study of over five hundred bills and the giving of testimony before the respective Committees in the House and Senate, fighting against the passage of bills detrimental to our profession and our patients and supporting those bills which would benefit "organized medicine" and improve health conditions throughout the nation. This AMA Legislative Committee performs a valuable service and should have the support of all our members. You can be of inestimable aid to this Committee in contacting your Congressman or Senator and requesting their help in supporting or opposing legislation as the case may be. You may be assured that your assistance is greatly needed and will be more than appreciated. You can obtain copies of bills and detailed information on federal medical legislation by writing the AMA Washington office, 1523 "L" Street, N. W., Washington 5, D. C. This is another of the many services our members receive, in addition to the Journal, for their twenty-five dollars.

AMA keeps records on all physicians. These records are started as soon as you enter a medical school and continue throughout your medical career, listing your birthplace, date of birth, the school from which you receive your M. D., the year of your graduation, whether or not you are a specialist, and your current home and office address. These records are of great help in compiling the "American Medical Directory".

Your Association encourages ethical medical practice. It has been said that one of the paramount reasons for founding the American Medical Association was to develop an accepted code of ethical conduct for physicians. The present AMA "Principles of Ethics" have been evolved through the years by action of the House of Delegates. They are interpreted by the Judicial Council, which serves as Medicine's Supreme Court.

This list of services which the AMA can and does offer you, as a member, has necessarily been condensed and is incomplete. Your Association, more or less, works in all areas of medicine, and can furnish you information in all fields pertaining to your profession, or can direct you to some other source, where such information can be obtained. You should remember that your Association is always eager and willing to help you in your practice.

Of course your membership entitles you to the further facilities of AMA in the use of medical, health films, exhibits for public or professional audiences, pamphlets to be used at fairs or public meetings, posters for school or industrial health education efforts, loans of radio transcriptions for broadcasting over local station, and TV films for use by Parish or District Societies over local television stations.

Dr. George Lull, Secretary and General Manager of the American Medical Association explained or portrayed the role a member plays in the work of the Association very well when he so aptly said that "an active, informed membership begins with your Parish Society, for the AMA is only as strong, only as progressive, only as active as the sum total of its component societies. It is in your Parish society that you

have your vote in organized medicine. It is through your Parish society that the medical profession can make itself felt as a group in your community life. Your role in organized medicine begins with the Parish Society."

It is equally important in this complex age that you be active in and informed about state and national medical affairs as well. Your AMA Journal provides you with a wealth of information. You can write either to your state society or to the AMA for still more information. Every member is always welcome at his state society office or at AMA headquarters. Visit them and find out for yourself. Attendance at state medical meetings is important, and a trip to an AMA meeting a unique experience. If you can tear yourself away from the maze of exhibits and other scientific attractions, visit your Association's House of Delegates. This body holds open meetings. Any member is welcome. Indeed, any member has the right and the privilege to appear and testify before any reference committee of the House.

Only if you are an informed, active member of the AMA will you benefit fully from its many services. And only then will AMA benefit from the suggestions, the ideas, and the service which you can contribute.

In order to continue its many services to the profession and the public, your Association needs your financial support. Of your $25.00 dues, $15.00 goes for your Journal subscription. Only ten dollars a year goes to pay for AMA's countless other activities. Compared to the dues received and the services rendered by other national organizations, you are getting a bargain. This is particularly true when it is remembered that for its first 100 years AMA received no membership dues at all!

You are the American Medical Association. You share in its prestige and its strength. You also must bear a share of the criticism it receives. Your Association asks from its members the loyalty to refrain from attacking it without knowledge; the loyalty of working from within to effect change rather than throwing rocks from without; the kind of loyalty that will make a member accept criticism of the AMA as criticism of himself, and, if the criticism is unjustified, defend his Association, or, if it is justified, take steps to correct the situation.

It should be well understood that, in order to participate in the many facilities and services accorded you as a member of the AMA you first have to become a member of your Parish or District, and State Societies.

We therefore urge and invite you to go the whole way with us, in joining up with your national association, thereby helping us to fight for the rights of our profession.

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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

MEDICAL EDUCATION SEEN AT CROSSROADS

America's medical schools face a crisis which can only be resolved by increased public interest and support, according to a 25-cent pamphlet, "The Challenge to Medical Education" just published by the Public Affairs Committee, 22 E. 38th Street, New York City.

Chancellor Henry T. Heald of New York University in an introduction calls attention to one of the main challenges faced by these "complex centers for training" by pointing out that "doctors are called up less and less to heal the sick and more and more to keep people well. Medical education must not only keep abreast of society's needs; it must anticipate them."

"In many ways, the medical establishment of our nation is stronger than it ever has been before," declares the author, Robert M. Cunningham, editor of *Modern Hospital*. "Every year,

more doctors care for more people, using more buildings, equipment, supplies, and helpers than in the year before. Year after year, new hospitals are built, offering their new facilities and their trained staffs for use by the medical profession.

"Nevertheless," he adds, "a threat to the nation's health does exist—the more serious because it lies hidden behind the spectacular medical achievements of our time and because it is a danger not so much to our present strength as to our future growth.

"The danger is that our great medical schools —the source of our supply of doctors and the medical advances of tomorrow—may not find the money and manpower to keep growing . . . as the body of our medical knowledge expands year after year," Mr. Cunningham asserts.

"By any estimate," he continues, "medical education is far more costly than education in most of the other professions . . . No other kind of education requires so much laboratory space and equipment and so much money to support the all-important dispensary and hospital services for clinical instruction."

Since the average medical student can afford to pay only about one-fifth of the cost of his training, the schools must find additional sources of support to offset the increasing costs resulting from modern scientific advances.

"Plainly, more and more groups must accept an increased share of responsibility for the support of medical education," Mr. Cunningham writes, "if we expect the schools to perform fully and effectively in the future."

The price of this pamphlet is 25¢ and may be obtained by contacting the Public Affairs Committee, 22 East 38th Street, New York City.

CLINICAL HEMATOLOGY COURSE

A three-day continuation course in Clinical Hematology will be presented at Tulane University beginning March 23, 1955. Dr. C. V. Moore, Dean and Professor of Medicine at Washington University, has accepted an invitation to be the guest speaker. Registration is now open to all physicians but will be closed March 1, 1955.

The program will be built around actual cases insofar as possible and will include discussion of the anemias, leukemias and lymphomas, polycythemia, purpura, problems of blood transfusion, and blood coagulation. Those interested should contact the Director of Graduate Medicine, 1430 Tulane Avenue, New Orleans, Louisiana.

BLAMES SEX HYSTERIA FOR DOUBLING ILLEGITIMATE BIRTHS AMONG 'TEEN-AGERS

The current wave of sex hysteria is bearing fruit in an increasing number of illegitimate births and a distressing problem of "bosom consciousness" among 'teen-agers, a Portland (Ore.) obstetrician and gynecologist reported at the Sixth Annual Congress on Obstetrics and Gynecology on December 14, 1954.

Speaking in the Palmer House before The Sixth American Congress on Obstetrics and Gynecology, Dr. Goodrich C. Schauffler said illegitimate births in the 'teen-age bracket have doubled in the last 15 years.

"This depressing fact is certainly significant of the general trend in relation to precocious sex activity," Dr. Schauffler said. "It must be recognized as a background to the observation that there is a greatly increased awareness of sex in the younger group, stimulated and maintained by sex hysteria, which is a calculated instrument of modern journalism and so-called entertainment trends.

RICHEST SOURCE OF VITAMIN C NOW READY FOR WORLD

A neglected backyard tree is about to give to the world its "richest known source of vitamin C" and to Puerto Rico a possible answer to its old one-crop economy problem.

After almost 10 years of efforts by scientists to interest "the giants of the food industry" in the potentialities of the "fantastic" acerola tree, the first commercial crop finally has been produced and is ready for the market.

The fruit is known as Puerto Rican cherry, West Indian cherry, acerola, or several other names. The three grows semi-wild in the Caribbean islands where natives say a tree in the back yard will "keep colds out of the front door."

And a six-ounce glass contains as much as 8650 milligrams of vitamin C—more than 85 times as much as a six ounce glass of fresh orange juice. It would take over 50 pounds of fresh raw cabbage to give the same amount of vitamin C as a glass of acerola juice.

The fruit is the size of an ordinary red cherry but is shaped more like a crab apple. Its taste is "tart and refreshing" and the tree bears four crops a year.

How the tree finally was developed commercially is told by Mrs. Anna May Wilson in the November Today's Health magazine, published by the American Medical Association. Her husband, Dr. James R. Wilson, director of the A.M.A.'s Council on Foods and Nutrition, was the man who interested orange juice millionaire Harvey Greenspan, Miami, in the acerola.

RISK TO EXPECTANT MOTHER WITH HEART DISEASE IS MINIMIZED

The outlook for the expectant mother with heart disease is most hopeful, a Minneapolis obstetrician reported at the Sixth Annual Congress on Obstetrics and Gynecology.

"With early appraisal, frequent examinations and vigorous medical care, the death rate can be

reduced to 1 per cent or less," Dr. Olga Hansen Litzenberg said at a symposium on medical complications of pregnancy before The Sixth American Congress on Obstetrics and Gynecology at the Palmer House.

The maternal mortality among women whose heart disease restricts their physical activities only slightly or not at all is only a little higher than in a comparable nonpregnant group, Dr. Litzenberg added. The death rate of infants is no greater, she said.

ASPIRIN CALLED BEST DRUG FOR MILD HEAD INJURIES

"Aspirin is the best drug for managing the ordinary restlessness, irritability and headache" which follow mild head injuries in children, according to Dr. Donald D. Matson, Boston neurosurgeon and assistant professor of surgery at Harvard Medical School.

When necessary, Dr. Matson writes in *Postgraduate Medicine* (3:16,1954), a physician may prescribe small amounts of Demerol or codeine to supplement aspirin. As a general rule, he cautions against the use of barbiturates.

Treatment of shock is a vital measure in all head injuries. The procedure usually effective in mild injuries includes: relief of pain with aspirin, simple warmth, reassurance and quiet. If treatment is carried out at home, parents should observe the child's alertness, motion of extremities and equality of pupils at set intervals.

PREGNANCY NO LONGER MORTALITY FACTOR IN POLIOMYELITIS

The pregnant poliomyelitis patient now has as good a chance for life as the non-pregnant polio victim, according to a report made at the Sixth Annual Congress on Obstetrics and Gynecology on Dec. 14, 1954.

Dr. Paula Horn of the University of Southern California School of Medicine, Los Angeles, said a five-year study at the Los Angeles County Hospital showed the death rate in pregnant adult women stricken with polio has now by modern care been cut to the same level as that of non-pregnant adult women—4 per cent in each instance. A few years ago the polio pregnant death rate was nearly 11 per cent.

Dr. Horn spoke in the Palmer House at the opening of the five-day Sixth American Congress on Obstetrics and Gynecology, sponsored by The American Committee on Maternal Welfare, Inc., and the American Academy of Obstetrics and Gynecology. The meeting was attended by about 3,500 physicians, nurses, public health officials and hospital administrators concerned with better care for mothers and babies.

The report covered 1,545 adult females stricken with polio in the five years, 1948-1952, inclusively. Of these, 225 women were pregnant. Nine deaths occurred in that group. There were 53 deaths among the 1,320 non-pregnant patients. The five-year mortality rate in each instance was 4 per cent.

In 1948, the first year of the study, there were five deaths in 65 pregnant women, or 7.6 per cent. Thirty-one deaths, or 7.1 per cent, occurred in 438 non-pregnant women. An earlier study, covering the years 1934-1947, inclusively, indicated pregnancy was a contributing factor to mortality, the rate being 10.9 per cent as against 2.3 per cent in non-pregnant women.

"Since 1948, there have been tremendous improvements in the management of bulbar spinal poliomyelitis," Dr. Horn reported. "Mortality appears to be related to the severity of the disease and not particularly to the trimester of pregnancy in which poliomyelitis was contracted. Mortality is no greater in pregnancy than in the non-pregnant women in the same age group.

"The management of a pregnant woman who contracts poliomyelitis in early pregnancy and continues to remain in a respirator is a prodigious task. This task has been made easier in recent years by the development of a positive pressure attachment to the respirator.

"With this, the respirator may be opened, and the patient can be kept breathing with a mask or endotracheal tube attachment. This greatly facilitates nursing care and certainly enables more adequate obstetrical examinations.

"It has been our experience that the first stage of labor in a respirator patient is 'relatively easy.' From the standpoint of prevention of respiratory depression, they have not been permitted barbiturates or narcotics during their illness. They thus appear to obtain sufficient relief in their labors from 50 mg. doses of demerol.

"Cesarean section can be safely performed in this critically ill group of pregnant women."

SOME PATIENTS DON'T WANT TO BE REASSURED

Sometimes the worst thing a doctor can do is to tell a patient he's perfectly well, a University of Virginia School of Medicine physician said recently.

Dr. Andrew D. Hart, Charlottesville, Va., said some patients don't really want to be reassured. These are the patients with "cardiac neurosis," which may cause real symptoms of heart disease but may only be a substitute or cover-up for greater problems—such as tension and nervousness or fear of mental illness.

He said this type of patient visits the doctor with complaints of heart symptoms, and is told firmly and reassuringly that he has no heart defect. The patient may then go to another, or more than one, doctor seeking another diagnosis. He may quote the first doctor as having said such things as "You have six months to live" or "I've never seen a worse heart than yours." The patient actually may believe this was what

the doctor said—since even normal memory is far from accurate, and nothing is so helpful as recollections to people who "dearly" need to believe something, Dr. Hart said.

ZEPHIRAN MOST EFFECTIVE IN TREATING RABIES WOUNDS

A 1 per cent solution of Zephiran chloride proved the most effective treatment of wounds in guinea pigs artificially contaminated with rabies virus, according to a report in the *Bulletin of the World Health Organization* (10:805, 1954).

Zephiran, a cationic detergent manufactured by Winthrop-Stearns Inc., was found to be the "material of choice" in comparison studies with fuming nitric acid and a soap solution. Persons bitten by a presumably rabid animal should also receive other approved prophylactics, such as anti-rabies vaccine or serum, state Drs. Howard J. Shaughnessy and Joseph Zichis of Chicago.

TEEN-AGE FATIGUE MAY BE PERFECTLY NORMAL

Teen-agers who are often tired and mope around the house may be ill—but are more likely to be quite normal, a New York physician said recently.

Dr. Edward A. Wilkes said teen-agers tire easily for one or more of a dozen reasons, and that most of the problems can be solved without expert help. He described the causes of fatigue in the November Today's Health, published by the American Medical Association.

The major reasons for normal fatigue are rapid growth, too little sunshine and outdoor exercise, social and school pressures with overstimulation and difficulty in sleeping, poor diet or overweight, and common teen-age emotional problems.

ARTIFICIAL VITAMINS

The American public in general is oversold on artificial vitamins, a New York University biologist declared recently.

Addressing the Rotary Club of Hoboken in the Grand Hotel, on November 9, Associate Professor Otto M. Helff of University College of Arts and Science at NYU said normal vitamin requirements could be filled by a wise diet at considerably reduced expense.

"Vitamin capsules and the like have an important place, but most of us get an overdose of vitamins when we buy them at the drug store," Professor Helff said.

"The best advice on vitamins comes from your doctor, and he should be followed precisely whenever vitamins are prescribed as a drug for arthritis, anemia, and other conditions.

"But people who buy vitamins that promise a week's supply of certain vitamin requirements in a one-day dosage are in many cases wasting six-sevenths of the money they pay. The reason is that the body cannot store more than one day's supply of certain vitamins."

EMOTIONAL FIRST AID NEEDED IN DISASTERS

You don't have to be a psychologist to give needed first aid to "emotional casualties" of a community disaster such as a flood, fire or tornado, said civil defense officials.

Knowledge of a few basic rules is all that's necessary to give first aid to a physically injured disaster victim. The same is true of persons who react badly to the emotional shock of a disaster, according to the Committee on Civil Defense of the American Psychiatric Association. Its material on psychological first aid, prepared for use in enemy attack but also helpful in natural disasters, appears in the Sept. 4 Journal of the American Medical Association.

The first need is to understand and control your own emotions and to know your abilities and limitations, so you will be in a position to help others. Then you must "accept every person's right to have his own feelings," however strange they may seem. Your job is to help the victim cope with his feelings, not to tell him how he should feel.

BOOK REVIEWS

On Burns; by Nathan A. Womack (ed.), Springfield, Illinois, Charles C Thomas, 1953, Pp. 179, $5.50.

This book consists of a rather complete and comprehensive coverage of the problem of the burned patient from the time of the initial injury to the time of discharge from the hospital. The salient features of the treatment of burns are presented through the medium of a panel discussion; the panel consisting of Dr. William Altemeier, Dr. James Barrett Brown, Dr. W. J. H. Butterfield, Dr. Truman Blocker, Dr. Julian Bruner, Dr. Stuart Cullen, Dr. Sam May, Dr. Carl Moyer, Dr. Sidney Ziffren.

In the rather informal discussions, which make up the book, one is able to read through the entire problem of burns and their management with a minimum of effort and didactic material. The comments at the end of each chapter are of particular interest since the floor is held open for discussion by all members of the panel.

In the first chapter, the results of the treatment of the burns at the University of Iowa hospitals are given by Dr. Sidney Ziffren. The most interesting part of the paper was the comparison by dividing the cases into two groups—the 1930 to 1940 group, and the 1940 to the 1950 group, significant differences being in the mortality and the incidence of infection and control of infection. The superior results in the second group were attributed mainly to the increased use of plasma and the plasma substitutes, as well

as whole blood and the antibiotics.

In the second chapter, Dr. Stuart Cullen discusses pain and its treatment. The principle stressed is that the burned patient does not necessarily need his routine dose of morphine and that extreme care and thought should be given in order to make the decision as to whether or not the patient requires sedation. It is pointed out that subcutaneous administration of morphine to a patient in shock or near shock can result in the medication not being absorbed, and therefore, the patient derives no benefit from it. Subsequent doses are then given and the shock treated. The result is, that when the shock is treated and circulation becomes adequate, intense narcosis may supervene. It was generally agreed by the panel that the best route was intravenous, so that the effect could be noted within fifteen minutes.

The open treatment of acute burns was thoroughly covered by Dr. Truman Blocker, who presented some statistics showing a decrease in the length of hospital stay, as well as in the incidence of infection. In addition, the need for intravenous therapy was markedly decreased.

Early care and grafting of acute burns was discussed by Dr. James Barrett Brown. The problem of infection and enzyme debridement of the burn or scar are discussed by Dr. William Altemier. Burn shock and its treatment and the use of the plasma substitutes are then discussed by Dr. W. J. H. Butterfield, and Dr. Carl Moyer.

The important points brought out in these chapters were that laboratory tests may not necessarily indicate the true status of the patient and the powers of observation should be used by the physician judiciously before instituting therapy; and then as a guard to the effectiveness of that therapy when instituted. It is recognized that at present we have no uniformity in satisfactory substitutes for plasma, and no method of stock-piling blood in case of an atomic attack. However, at the present time although some hyperpyrexia has been reported with its use, apparently, dextran is the plasma expander of choice.

The treatment of the late burn wound and its contractures was next presented by Dr. James Barrett Brown. The use of split thickness grafts, as well as pedicle coverage are discussed.

Adrenal cortex function and severe burns are discussed by Dr. W. J. H. Butterfield, the nutrition of the burn patient by Dr. Truman Blocker and finally delayed coverage of the burn wound and joint motion is discussed by Dr. Julian Bruner. Dr. Bruner deplores the fact that so many burns leave the hospital with joints which are hopelessly limited in motion and for which physiotherapy offers all too little from a standpoint of restoring normal function.

The book ends with a general discussion by all of the members of the panel.

WILLIAM J. CHAMPION, M. D.

A Dynamic Psychopathology of Childhood, by Lauretta Bender, Springfield, Illinois, Charles C Thomas, 1954, Pp. 275, Price $7.50.

This is the third book of the Bellevue Studies of Child Psychiatry and is a collection of papers written by Lauretta Bender, Paul Schilder, and their associates, who have been examining, treating, and observing children for the last quarter century in the Psychiatric Division of Bellevue Hospital. It deals with various psychopathological responses in children and considers their effect on subsequent development of personality. Ninety case reports of children with follow-up social histories are presented, along with additional observations on two hundred children. The topics treated are development of language and the symbol, hallucinations, imaginary companions, body concepts, impulsions, compulsions, obsessions, sexual expression of identification conflicts, reactions to death and war, comic books, and the ideology of children. The discussion of each of these topics is general and based primarily on clinical observations. The book should be of interest to professional persons whose work brings them in contact with children, whether these children be emotionally disturbed or well adjusted.

NANCY SIMMS

Lectures on General Pathology, by Sir Howard Florey (ed.), Philadelphia Pa., W. B. Saunders, 1954, Pp. 733, Price $13.00.

This new British book, as the title indicates, is concerned with a variety of topics in the field of general pathology in contradistinction to the pathology of particular organs. It consists of 37 chapters, more or less in lecture style by 10 contributors, 8 of whom are at Oxford. In addition to the customary subjects of inflammation, primary tissue changes, immunity, pathogenicity of microbes and healing, there are other chapters ranging from fever and edema to the biologic effects of radiation.

Reviewing this book has been a pleasure. It is a delightful blending of the old and the new. Most of the chapters could be read with little effort and were notably free of "literary road blocks." Where the path was rougher, as in the chapter dealing with antigen-antibody reactions, it could probably be attributed to the subject matter. The references at the end of each chapter are very adequate and carefully chosen. It is regrettable that every contributor did not follow the practice of R. G. MacFarlane in indicating briefly the content of each article referred to. Some teachers who use the book may wish that some of the subjects had been omitted and others treated more extensively. It is heartily recommended to all medical students, who should purchase it at the end of their freshman year. The graduate physician of ten years or more will also find much here that he has forgotten and more that is new.

Only $13.00 stands between this reviewer and a copy on his shelf.

E. B. FERGUSON, JR., M. D.

The Digital Circulation, by Milton Mendlowitz, New York, Grune & Stratton, 1954, Pp. 182, Price $6.75.

This monograph on the circulation to the digit is written by one who has devoted a considerable part of his research and clinical time to the study of the peripheral circulation and, in particular, that of the digits of man. His monograph is a fairly comprehensive review of the anatomy, physiology, pharmacology, pathology, methods of study, and disease states of the digital blood vessels. The author has presented well these phases of the peripheral circulation. The only adverse criticism is his failure to have included more, to have been more critical of the work in the literature, and present further his own impressions of the studies of others for the benefit of readers who are less trained in this field. Although it is evident that it was not his intention to cover the subject completely, the material presented could have been evaluated more critically.

Even though, Mendlowitz has concerned himself with the calorometric method for digital blood flow, he presents other procedures employed in the study of the digital circulation. The methods are not described in detail but are well illustrated so as to make readily possible the understanding of the various methods and their relative merits and disadvantages. Surely, anyone embarking upon any studies of the digital circulation would find this monograph useful for orientation purposes as an adjunct to a critical review of the methods of study. Such a review would also be aided by the extensive bibliography included at the end of the book.

Various disease states are discussed briefly. Normal physiologic reactions are also described, including hemodynamic concepts pertaining to the peripheral circulation. It must be remembered, however, for example, that the values for resistance in the vessels of the finger tip are only approximations based upon the limited available objective data. These attempts at precise definitions of the hemodynamic states are important, even though the errors may be large, if the role of the digital circulation in the entire cardiovascular adjustments are to be appreciated.

The book is clearly written and well illustrated throughout and is recommended to those who are interested in the normal and abnormal circulation of man. Obviously, as any monograph, this one is not complete, as is evidenced by the author's attempt to condense his own experience, knowledge, and the contents of 636 publications into 146 pages. Mendlowitz has succeeded in eliminating much of the useless material contained in the medical literature for the benefit of the readers whose time for detailed study of the literature may be limited.

G. E. BURCH, M. D.

The Physician and his Practise, Edited by Joseph Garland, M. D., Boston, Mass., Little, Brown & Co., 1954, Pp. 270, Price $5.00.

This book is a collection of articles by eighteen authorities. The various relationships of the physician as regards his family, his neighbor, his patient and his community are presented briefly, clearly and with apparent first-hand knowledge. The business aspects of medical practise in its several forms are well done. Rather than a rigid blue print of proper procedure it attempts a broad approach and succeeds quite well. It is a safe guide for those seeking a way on the threshold of the practise of medicine.

I. L. ROBBINS, M. D.

Acute Anuria, by Claus Brun. Copenhagen, Ejnar Munksgaard, 1954. Illus. Pp. 215.

This is a 215-page monograph on the acute anuric syndrome by a well known Danish clinical investigator. It is based upon a personal study of 32 patients. In many of these cases, both detailed functional studies and renal biopsies were carried out, and upon this depends the book's particular merit. The author prefers the term "acute tubulo-interstitial nephritis" to other designations of the kidney lesions. Of special interest were two cases which showed a definite oliguria and renal functional changes of moderate severity with only the slightest histologic changes in the biopsy specimens. In the reviewer's opinion the book will be most useful in providing detailed knowledge of the pattern and range of renal functional disturbance encountered in various examples of the syndrome. For this and other reasons it is recommended to students and physicians.

E. B. FERGUSON, JR., M. D.

PUBLICATIONS RECEIVED

W. B. Saunders Co., Phila.: Pediatric Diagnosis, by Morris Green, M. D., and Julius B. Richmond, M. D.; Standard Values in Nutrition and Metabolism, edited by Errett C. Albritton, A. B., M. D.

Charles C Thomas, Publisher, Springfield, Ill.: Hysterectomy, by John C. Burch, M. D., and Horace T. Lavely, M. D.; Surgery of the Elbow, by Frederick M. Smith, M. D.; The Human Brain in Sagittal Section, by Marcus Singer, Ph.D., and Paul I. Yakovlev, M. D.; Hernia, by Chester B. McVay, M. D., Ph.D.; Vaginal Hysterectomy, by Laman A. Gray, M. D.; Hemorrhage of Late Pregnancy, by John S. Fish, M. D., R. A. Bartholomew, M. D., E. D. Colvin, M. D., W. H. Grimes, Jr., M. D., and W. M. Lester, M. D.; Diabetes Mellitus, Objectives and Methods of Treatment, by Henry T. Ricketts, M. D.